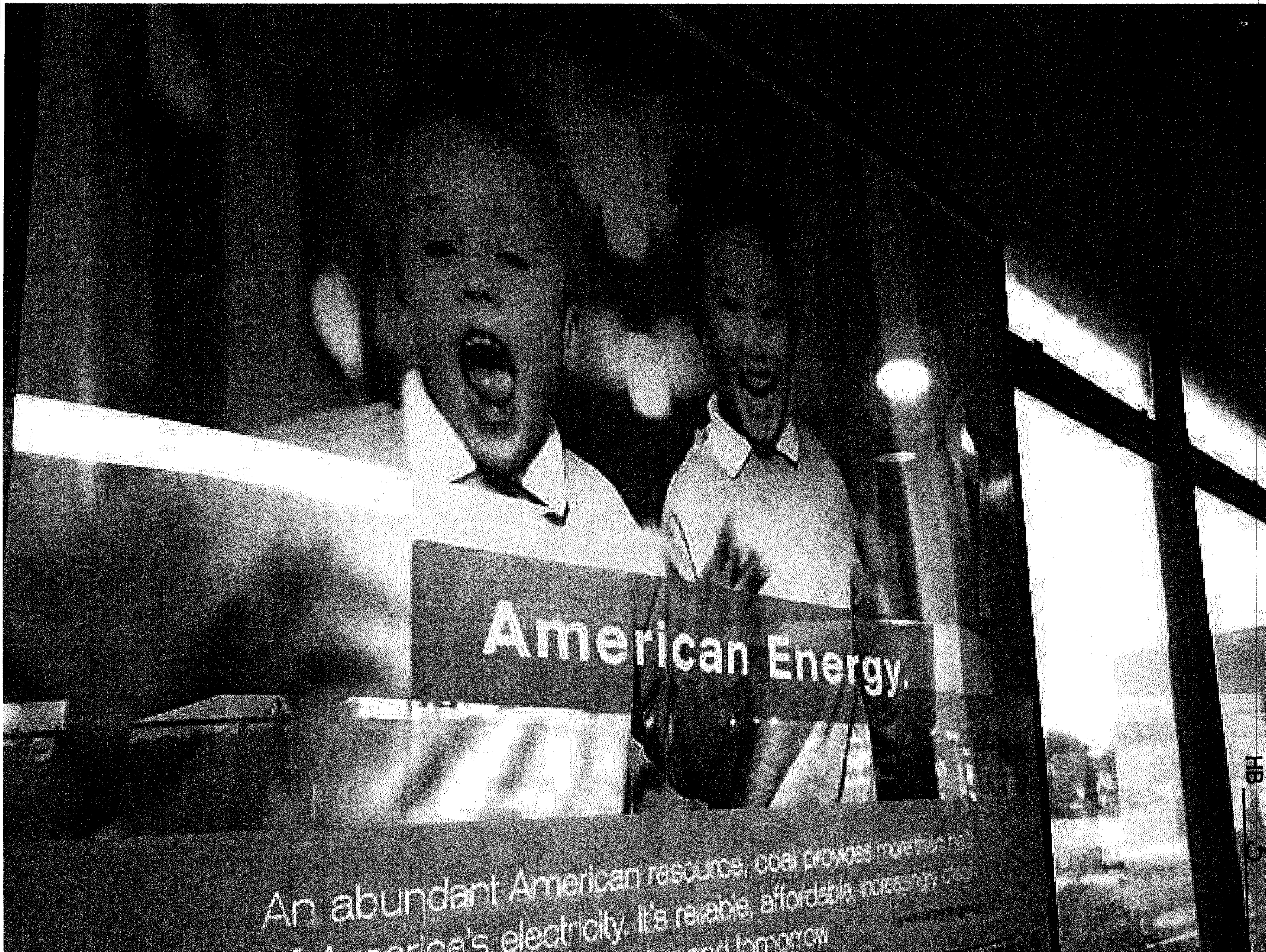


EXHIBIT 5
DATE 2-19-07
HB 5


American Energy.

Coal-Related Activities for Elementary Students

AMERICAN COAL FOUNDATION

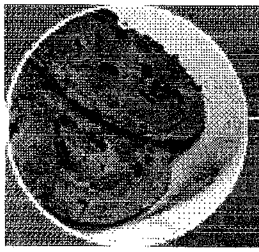
101 Constitution Avenue N.W., Suite 525-E
Washington, DC 20001-2133
202/463-9785 • 202/463-9786 fax • info@teachcoal.org
www.teachcoal.org

Coal-Related Activities for Secondary Students

AMERICAN COAL FOUNDATION
101 Constitution Avenue N.W., Suite 525-E
Washington, DC 20001-2133
202/463-9785 • 202/463-9786 fax • info@teachcoal.org
www.teachcoal.org

AMERICAN COAL FOUNDATION

Lesson Plans



Grade Level 3-8

Cookie Mining

Overview:
Students participate in a simulation of the mining process using chocolate chip cookies and toothpicks. The simulation helps to illustrate the costs associated with the mining of coal.

Objectives:
Students will:

1. participate in a simulated "mining" of chocolate chips from cookies, using play money to purchase the necessary property, tools, and labor;
2. understand the various costs associated with mining coal, including environmental remediation, as demonstrated in the simulation; and
3. calculate costs and profits from cookie mining and relate them to the mining industry.

National Standards:
National Council for the Social Studies (NCSS) Standards

- Production, Distribution, and Consumption
- Science, Technology, and Society

National Council of Teachers of Mathematics (NCTM) Standards

- Numbers and Operations, 3-5

Time Needed:
One to two class periods

Materials:

- Play Money
- Three different types of commercially packaged chocolate chip cookies
- Grid paper
- Pencils

- Flat toothpicks
- Round toothpicks
- Paper clips
- **Cookie Mining Worksheet**

Discussion Questions:
What do you think are some of the costs associated with mining coal?
Do you know what the term land reclamation means?
If not, what do you think it might mean with regard to coal mining?

Procedure:

1. Review the costs associated with coal mining: land acquisition, labor, equipment, and reclamation. Coal companies are required by federal law to return the land they mine to its original, or an improved, condition. This process, known as reclamation, is a significant expense for the industry.

2. Explain that the mining industry, like any other business, faces challenges to make itself profitable. To understand some of these challenges, students will attempt to conduct a profitable mining business in an experiment that requires them to mine the "coal" chips from chocolate chip cookies.

3. Give each student $19 in play money, a sheet of grid paper, and a **Cookie Mining Worksheet**. Allow each student to purchase one "mining property" (a cookie) from three separate brands available. Montana costs $3, Pennsylvania costs $5, and Kentucky costs $7. Students may want to examine the cookies before deciding which one to purchase.

4. Once all the students have purchased their property, have them measure it by placing it on the grid paper and tracing it. Then have them count the number of squares that fall inside the circle (partial squares count as full squares). Tell students to record this number on the **Cookie Mining Worksheet** under *D. Reclamation*.

5. Have each student purchase "mining equipment" (flat and round toothpicks and paper clips). More than one piece of equipment may be purchased, but no tools may be shared among students. Sell a flat toothpick for $2, a round toothpick for $4, and a paper clip for $6. Sell replacement tools when necessary.

6. Explain that each minute of mining (labor) costs $1 and that each chocolate chip mined from their property will result in a $2 profit. Broken chips may be combined to form a whole chip. Consumed chips will eat into profits!

7. Do not allow students to spend more than five minutes mining. If they spend less time, their labor cost will be lower. Have them record their mining time and labor cost under *C. Mining/excavation costs* on the **Cookie Mining Worksheet**.

8. After everyone is finished mining, have students restore their property to its original condition, within the drawn circle on the grid paper. This "reclamation" should also be timed, (no more than three minutes) and students may only use their tools, not fingers. After time is up, collect additional reclamation costs ($1) for each square covered outside the original outline. Disburse profits for chips mined. Have students use the **Cookie Mining Worksheet** to calculate their profit or loss.

Assessment:
Allow students to share their experiences with the class. Was making a profit easier or harder than they expected? How accurate is this simulation in illustrating the challenges of making money in the mining industry? What costs or possibilities for profits were not included in this exercise?

Extension:
Encourage students to design another profit/loss simulation for a different industry. Remind them to think of all the costs related to the industry and to try to create an exercise that can be done in a short period of time by the rest of the class. Have them prepare a worksheet for other students to complete after participating in the simulation, on which to calculate their profit or loss.

Differentiation:
Working in tandem to complete the profit/loss worksheet might be helpful for those with math-related learning disabilities.

Was this lesson helpful? | **Printing our lesson plans.**

About Us | **All About Coal** | **Lesson Plans** | **Resources** | **Glossary** | **Teacher Store** | **News & Events** | **Con**

AMERICAN COAL FOUNDATION

All About Coal

Coal and the Environment: Land and Air

Although abundant and cost-effective, coal has some environmental challenges to overcome. Coal has a reputatio dirty fuel source, one that can pollute the air and damage the landscape.

So that coal might live up to its potential as a significant source of energy, the coal industry is working hard to ensu the mining and use of coal does not permanently damage land or pollute air. The coal industry restores mined land original condition or prepares it for more productive uses, and it has incorporated technological advances to keep t clean and safe.

Reclaiming Land

In the past, coal mining often left behind landscapes that were unattractive and unproductive. Animals and plant lif once thrived in an area could no longer survive in conditions produced by coal mining. Today, thanks to land recla can be difficult to tell the difference between land that has been mined and land that has not.

Land reclamation is the process of protecting, restoring, or improving land before, during, and after surface mining. means that the land is preserved, nature is protected, and water and soil are conserved. Ultimately, the land can b productive farmland, be restored to forest, or undergo development as a lake.

How does this work?

First, geologists study rock formations to find possible coal reserves. Test holes are drilled so that samples of coal examined for evidence of quality, quantity, and location beneath the surface.

Then, the reclamation plan is made. This involves studying the area's geology, plants, water, soil, and wildlife. Info recorded so that potential problems during the mining process can be identified and so that conditions may be rest the mining has been completed. In addition, the mining site is examined for artifacts from past cultures. If any are f they are removed and preserved.

Next, the timeline for mining and reclamation is set. The coal mining company works with federal and state govern agencies and local officials to ensure that all regulations are being followed.

Once the planning phase has been completed and the required permits obtained, mining may begin. The topsoil, s earth, and rock are removed and set aside, and the coal that lies just below the surface is mined.

During the mining process, water is tested to ensure that it is not polluted. If water is found to be contaminated, it is before it leaves the mine area.

Finally, the area is refilled with the overburden (the earth and rock that were removed) and soils. To the extent pos area is restored to its original condition or improved. This often means planting, seeding, and irrigating the land—a completed over many years.

Reducing Air Pollution

When coal is burned, it releases impurities such as sulfur, nitrogen, and fly ash. These can pollute the air or contrib conditions that can lead to "acid rain," precipitation that damages forests and pollutes rivers and lakes. Today, thou advances in technology mean that about 99 percent of the chemicals that can pollute the air and more than 95 per the chemicals that can cause acid rain are removed.

These technological advances are part of an effort referred to as the Clean Coal Technology (CCT) Program, whic in 1985. Since that time, the federal government has contributed more than $2 billion toward this program to make burning of coal cleaner and safer. The coal industry has contributed more than $4 billion toward this goal.

How is coal made cleaner?

There are several ways. Coal can be crushed and washed before it is burned. The washing process often goes a l in removing harmful sulfur, but it does not remove all of the sulfur.

While the coal burns, special combustion processes can remove more sulfur and nitrogen. Devices called *flue gas desulfurization systems*, or "scrubbers," remove more than 90 percent of the sulfur dioxide emissions from the burr process. The flue gas is sprayed with a mixture of water and lime or limestone, which reacts with the sulfur dioxide wet sludge or, in some cases, a dry powder that can be disposed of or made into pellets for roadbeds or into plaste concrete blocks.

Another method for tackling air pollution involves using devices called *electrostatic precipitators*, which give coal du particles an electric charge so they can be attracted to a collector plate.

Other methods of removing pollutants involve the way the coal is actually burned. In *fluidized bed combustion* (FBC inserted into a bed of particles (including limestone) that are suspended in the air and react with the coal to heat th more cleanly. In FBC, coal is burned at a slightly lower temperature, which helps prevent some nitrogen oxide gase forming. The result is that FBC can remove more than 90 percent of the sulfur and nitrogen while the coal is burnin Through a chemical reaction, sulfur gases are changed into a dry powder called calcium sulfate, which can be use wallboard for building homes.

The *coal gasification* method changes coal into a gas that has the same heating value as natural gas. Coal gas bu cleaner than coal because so many pollutants have been removed during the transformation to a gas. This methoc remove up to 99.9 percent of the sulfur and tiny dirt particles from burning coal. For more information about coal ga go to: http://www.eastman.com/Company/Gasification/Overview.htm.

Was this article helpful?

The Coal Bowl

OBJECTIVE:

Students will review facts about coal use and its importance in the United States.

CONCEPTS:

- Coal is used to produce electricity, to provide heat for industrial and retail manufacturing processes, in iron and steel production and for export.
- Coal is present in 38 states, and is widely distributed and used across the United States.
- The four major types of coal are: lignite, sub-bituminous, bituminous and anthracite.

SKILL REINFORCEMENT:

Critical thinking
Math-number manipulation
Cooperative learning

GRADE LEVEL:

4–8

TIME NEEDED:

One class period or less

MATERIALS:

- worksheet
- list of categories
- teacher answer sheet
- play money

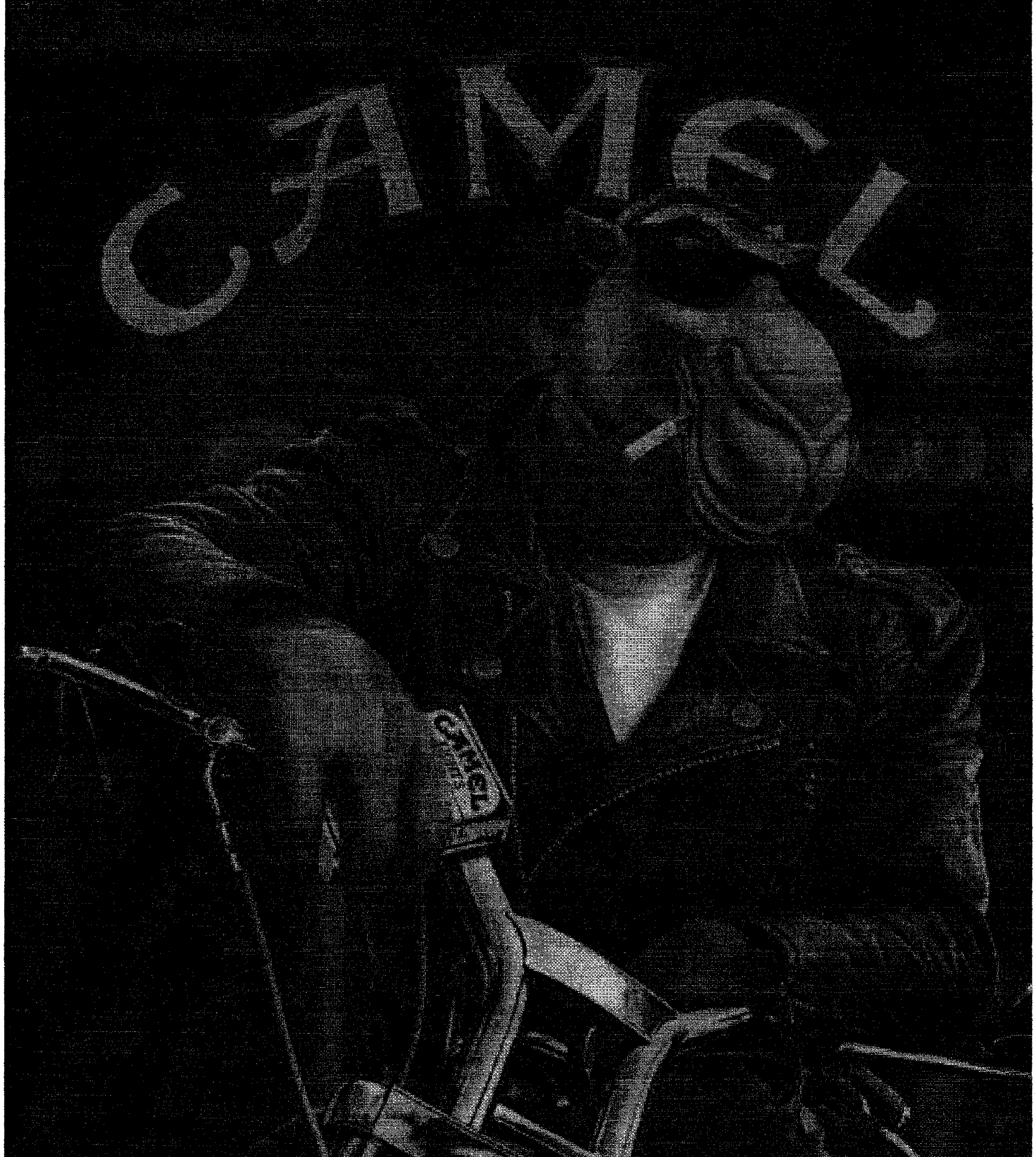
PROCEDURE:

After students have studied about coal, they can take this Coal Bowl.

1. Place class into teams.
2. Put The Coal Bowl Categories on the blackboard or an easel for class viewing.
3. Students, using acquired knowledge and resource aids, complete the Coal Bowl Worksheet. Place an appropriate time limit on this (about 15 minutes).
4. Teacher calls out a Coal Bowl Category and asks the first team to raise its hand to answer. The team is rewarded with one "Coal Dough" for each correct answer in that category. Call on another team as necessary to finish correct answers.
5. Repeat until quiz is completed. The team with the most "Coal Dough" is the winner.
6. Review categories that seemed to stump most students.

ACTIVITY DEVELOPED BY:

Kathryn McCoy, Tennessee Teacher


CAMEL
CAMEL

Coal Dough

$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1

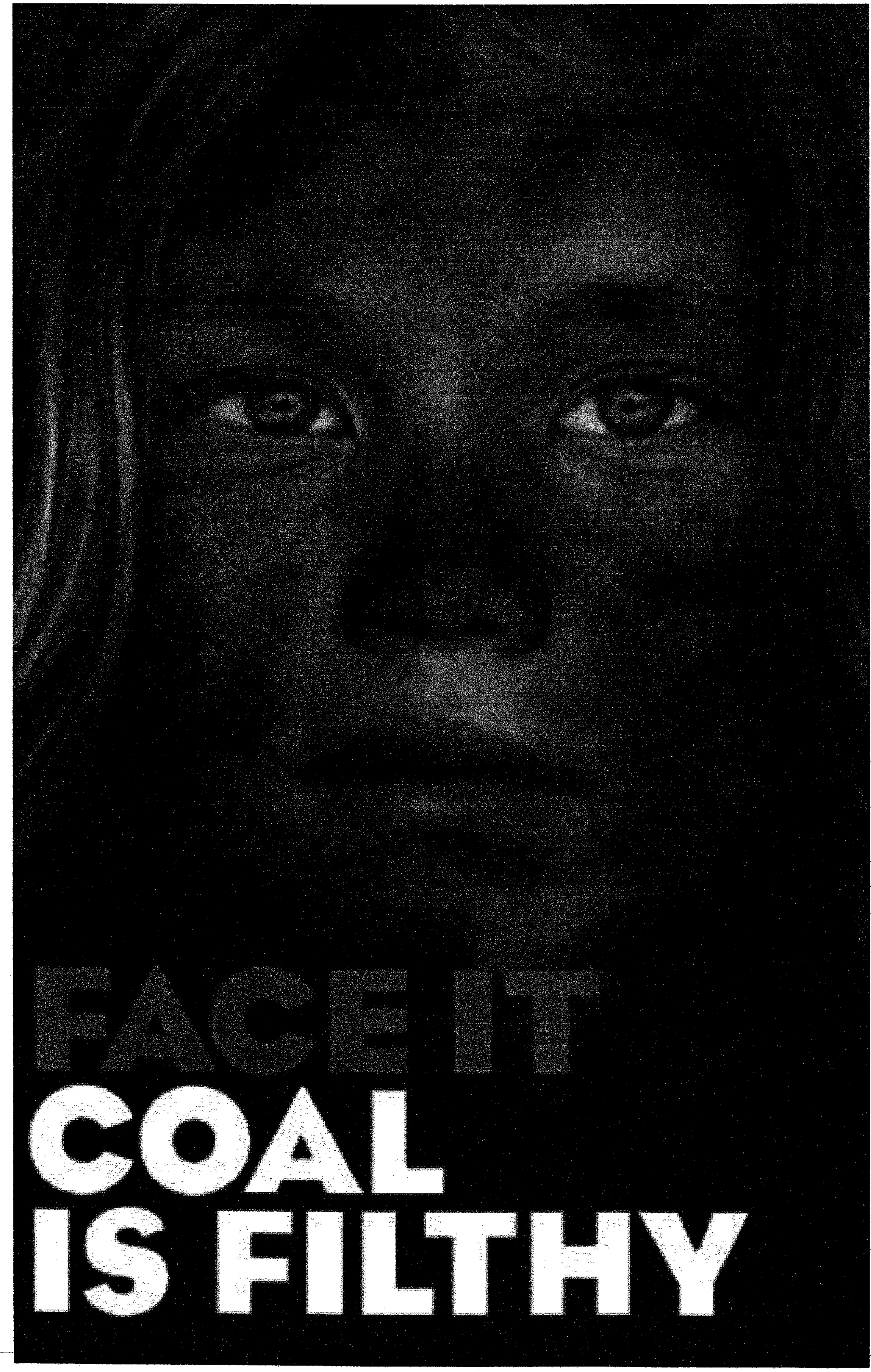
$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1

$1 $1
COAL
DOUGH
$1 $1


Smooth
reaper.


FACE IT
COAL
IS FILTHY

TC ONLINE

Development of a model of the tobacco industry's interference with tobacco control programmes

W M K Trochim, F A Stillman, P I Clark and C L Schmitt

Tob. Control 2003;12;140-147
doi:10.1136/tc.12.2.140

RESEARCH PAPER

Development of a model of the tobacco industry's interference with tobacco control programmes

W M K Trochim, F A Stillman, P I Clark, C L Schmitt

Tobacco Control 2003;**12**:140–147

See end of article for authors' affiliations

Correspondence to: William M Trochim, 249 MVR Hall, Department of Policy Analysis & Management, Cornell University, Ithaca, NY 14853, USA; wmt1@cornell.edu

Received 2 January 2003. Accepted 23 January 2003

Objective: To construct a conceptual model of tobacco industry tactics to undermine tobacco control programmes for the purposes of: (1) developing measures to evaluate industry tactics, (2) improving tobacco control planning, and (3) supplementing current or future frameworks used to classify and analyse tobacco industry documents.

Design: Web based concept mapping was conducted, including expert brainstorming, sorting, and rating of statements describing industry tactics. Statistical analyses used multidimensional scaling and cluster analysis. Interpretation of the resulting maps was accomplished by an expert panel during a face-to-face meeting.

Subjects: 34 experts, selected because of their previous encounters with industry resistance or because of their research into industry tactics, took part in some or all phases of the project.

Results: Maps with eight non-overlapping clusters in two dimensional space were developed, with importance ratings of the statements and clusters. Cluster and quadrant labels were agreed upon by the experts.

Conclusions: The conceptual maps summarise the tactics used by the industry and their relationships to each other, and suggest a possible hierarchy for measures that can be used in statistical modelling of industry tactics and for review of industry documents. Finally, the maps enable hypothesis of a likely progression of industry reactions as public health programmes become more successful, and therefore more threatening to industry profits.

A substantial peer reviewed literature exists describing the great variety of strategies and tactics the tobacco industry uses to undermine public health. A good deal of this work has documented, at least qualitatively, the tobacco industry's specific actions to prevent or undermine tobacco control programmes and organisations.[1–10] The tobacco industry has been concerned that large scale, comprehensive tobacco control programmes would reduce smoking and thus reduce profits.[11 12]

A prime example of a programme that the industry perceived as a threat was the American Stop Smoking Intervention Study (ASSIST)[13 14] which was the first, large multi-state initiative (1991 to 1999) that sought to reduce tobacco use by changing the sociopolitical environment through media and policy advocacy, and the development of state infrastructure to deliver tobacco control.[15] Given its scope, it is not surprising that ASSIST caught the attention of the tobacco industry. For example, Andrew H Tisch, then chairman and CEO of Lorillard Tobacco Company, delivered a speech in 1992 that described how threatening the ASSIST programme was to the industry.[16]

A major purpose of the ASSIST project was the evaluation of its effects. Detailed measures were collected on both the programmes (including the capacity, resources, and efforts involved in implementing the various programme components) and outcomes (both intermediate and long term). However, because of the presence of the industry, tobacco control programmes cannot be evaluated like most other programmes. While local, state, and federal governments are expending resources to reduce smoking rates and promote tobacco control, the tobacco industry is expending significant resources to promote sales of their product, influence governments, and undermine these programmes. The industry's anti-tobacco control efforts constitute a countervailing force to tobacco control programmes that needs to be considered when evaluating programme effectiveness since industry efforts could actually swamp any impact coming from these programmes, reduce measurable outcomes, and lead to an underestimation and devaluation of the impact and effectiveness of tobacco control efforts.

The ASSIST evaluation was the first major tobacco control evaluation to hypothesise a relationship between the industry's anti-tobacco control efforts and the programme.[17] ASSIST included the construct of pro-tobacco efforts in the overall evaluation model (fig 1). However, before this construct can be operationalised, it needs to be conceptualised well. Categorising the dimensions of anti-tobacco control tactics and building a comprehensive model of these actions is a necessary first step toward development of measurable components and indices that can be used in programme evaluation. While originating in connection with the ASSIST initiative, this problem of accounting for industry counter-efforts is not limited to that context alone, but is of relevance in the evaluation of any tobacco control programme.

Currently, there is no overarching conceptual model that could guide operationalisation of measures of industry tactics that might be useful for evaluation. Outside of the informal and anecdotal literature on specific industry tactics, about the closest thing to a current standardised framework that might be applicable for describing industry tactics is the UCSF/ANRF Tobacco Documents Thesaurus, a detailed glossary of terms used to index tobacco industry documents.[18] However, the Thesaurus was not designed to provide a conceptual framework for tobacco industry tactics. It is essentially a vocabulary of standard subject terms, or keywords, used to index and describe all documents in the tobacco control field.[19 20] While essential for document research, it has little utility for operationalising measures of industry tactics.

Abbreviations: ASSIST, American Stop Smoking Intervention Study; MDS, multidimensional scaling; UCSF/ANRF, University of California San Francisco/American Nonsmokers Rights Foundation

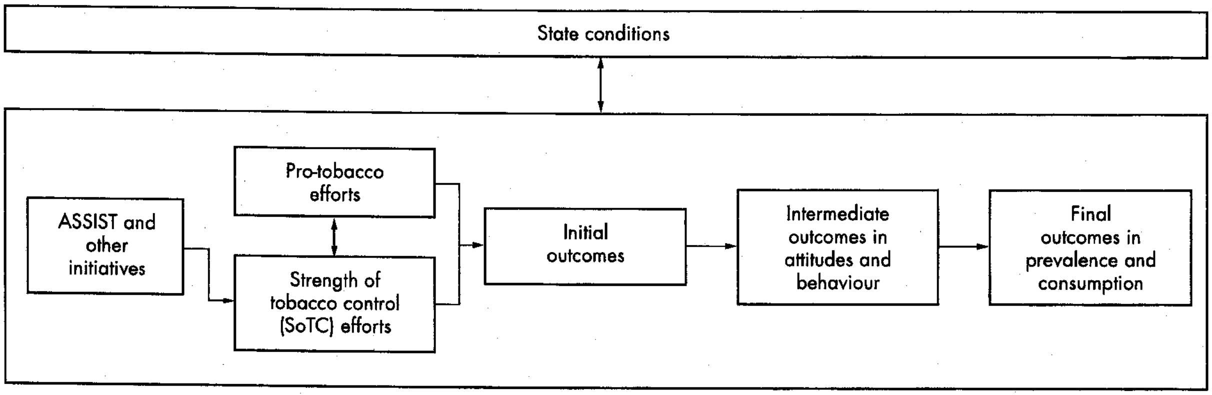


Figure 1 General conceptual model for the ASSIST evaluation.[17]

This paper describes the development of a comprehensive conceptual map of the tactics that the tobacco industry uses to undermine tobacco control efforts. The resulting conceptual map, developed in the context of the ASSIST evaluation, has utility beyond that context for the development of measures for programme evaluation, for improving strategic level tobacco control programme planning, and for informing current or future frameworks used to classify and analyse tobacco industry documents.

METHODS

The concept mapping methodology[21] was used to develop the conceptual model of pro-tobacco tactics. Concept mapping is a participatory mixed methods approach that integrates group process activities (brainstorming, unstructured pile sorting, and rating of the brainstormed items) with several multivariate statistical analyses (multidimensional scaling and hierarchical cluster analysis) to yield both statistical and graphic representations of a conceptual domain.

Participants

The participants were selected because they had previously encountered overt industry resistance to tobacco control programming and/or research, had published research arising from searches of the industry documents, or had otherwise demonstrated understanding of industry challenges to tobacco control. Among those represented, all were from the USA, 15 were academics, seven represented advocacy organisations, seven contract research organisations, four government agencies, and five were from tobacco control funding organisations (classifications not mutually exclusive). All participants (n=34) utilised a web based program* to participate in the mapping process (brainstorming, or sorting and rating, or both). A subset of this group (n=13) participated in a face-to-face expert panel to interpret the results of the electronic mapping process.

Procedures

The general procedure for concept mapping is described in detail elsewhere.[21] There were four distinct phases in the process: brainstorming, sorting and rating, data analyses and generation of the maps, and expert panel interpretation of the maps.

Brainstorming

The experts logged on to a private web page over a four week period. Each brainstormed statement was generated in response to the prompt: "One specific activity/tactic the tobacco industry uses to oppose tobacco control is . . ." They entered the statements in a list without regard to structure, hierarchy, or clustering of statements. The process resulted in generation of 226 statements.

In preparation for the sorting and rating task, the 226 statements were edited and consolidated. The process used was one of grouping statements that were similar, then constructing one statement that captured the content of the group of statements. The goal was to have a set of mutually exclusive statements, with only one main idea in each, and with no loss of content from the original list. In this manner, the original 226 statements were consolidated into the final set of 88 statements.†

Sorting and rating

Twenty one of the experts were asked to log on to another web page for the sorting and rating tasks. Each conducted an unstructured sorting of the statements.[22–24] They grouped the brainstormed statements into piles "in a way that makes sense to you". The only restrictions in this sorting task were that each statement could not be its own pile, there could not be a pile consisting of all the statements, and there could be no "miscellaneous" pile (any item thought to be unique was to be put into its own pile). Each expert was asked to supply a brief label that summarised the contents of each of their groups/piles.

Each participant was then asked to rate the 88 statements with these instructions: "Rate each statement on a 1 to 5 scale for its relative importance in undermining tobacco control efforts. Use a 1 if the statement is relatively unimportant (compared to the rest of the statements) in undermining tobacco control efforts; use a 5 if it is extremely important. Although every statement probably has some importance (or it wouldn't have been brainstormed), try to spread out your ratings and use each of the five rating values at least several times."

Data analyses and generation of the maps

The analyses‡ began with construction from the sort information of a binary, symmetric matrix of similarities. For any two items, a 1 was assigned if the two items were placed in the same pile by the participant, otherwise a 0 was

*The Concept System Global® web software was used for all web processes on this project. Further information on the software may be obtained from Concept Systems Inc, http://www.conceptsystems.com/

†Detailed and intermediate results, including the original list of 226 brainstormed statements, can be obtained at http://omni.cornell.edu/tactics/

‡All analyses were accomplished and results produced using the Concept System software, version 1.75. Further information on the software may be obtained from Concept Systems Inc, http://www.conceptsystems.com/

Table 1 Statement numbers, statements within clusters listed in descending order of average importance, and importance rating mean and standard deviation (SD)

Number	Statement	Mean	SD
Lobbying and legislative strategy		**3.71**	**0.94**
85	Writing and pushing pre-emptive legislation at state level	4.67	0.58
8	Creating loopholes in laws and agreements (e.g. the MSA) to allow business as usual	4.57	0.68
26	Contributing funds to political groups at federal, state and local level, to support industry goals	4.43	0.98
53	Using clout to influence introduction, advancement, modification, or suppression of bills in legislative bodies	4.38	0.74
87	Lobbying to assure that funds directed to tobacco control are diverted to non-tobacco control initiatives	4.33	0.73
27	Using clout to limit powers of regulatory agencies (jurisdiction, procedures, budgets)	4.29	0.78
63	Providing legislators with contributions, gifts, and other perks	4.10	0.77
44	Promoting partial or weak measures as an alternative to effective measures	4.10	0.77
52	Inserting limiting language in legislation, such as "knowingly" sell tobacco to minors	4.05	0.74
13	Writing weak tobacco control legislation then arguing that tobacco control measures are ineffective	3.86	0.85
17	Ghost writing non-tobacco bills (e.g. sewage) with clauses that if enacted, would bring pre-emption via the backdoor	3.71	0.90
7	Lobbying government officials to set unrealistic tobacco control goals to ensure programme failure	3.67	1.20
61	Using political and/or monetary clout to delay funding of tobacco control programmes	3.67	1.06
36	Lobby to assure that funds are diverted to ineffective tobacco control activities	3.67	1.06
62	Working against campaign finance reform to maintain influence	3.62	1.12
21	Working against strengthening campaign and lobbying disclosure laws	3.57	1.08
19	Promoting tort reform	3.38	1.24
41	Using clout to assign tobacco control programmes to hostile/apathetic agencies for implementation	3.19	1.08
76	Conducting "briefings" of members of Congress, allies, and consultants to sway opinion on an issue	3.14	1.06
1	Promoting smokers' rights legislation	3.05	1.02
29	Use of tobacco companies subsidiaries (i.e. Miller and Kraft) in political opposition to tobacco control legislation	3.05	1.12
10	Ensuring supportive legislators will lob soft questions during testimony	2.38	0.92
2	Using tobacco employees to lobby against legislation with the excuse that it threatens their job security	2.38	1.16
Legal and economic intimidation		**3.46**	**1.04**
16	Devoting considerable resources to legal fights	4.76	0.44
65	Create and fund front groups	3.81	1.12
46	Assuring that court battles are fought in favourable jurisdictions	3.76	0.83
64	Infiltrating official and de facto regulatory organisations (like ASHRAE)	3.43	1.16
58	Filtering documentation through their attorneys in order to hide behind attorney work product	3.29	1.35
9	Encourage (or fail to discourage) smuggling as a way to counter tax hikes.	3.10	1.26
4	Counter tax increases with promotions and cents off	3.05	1.20
48	Threatening to withdraw support from credible groups to control	2.48	0.98
Usurping the agenda		**3.39**	**1.12**
42	Developing alliances with retailers, vendors, and the hospitality industry in opposition to public health policies	3.90	0.89
40	Usurping the public health process, such as creating their own youth tobacco prevention programmes	3.33	1.20
22	Avoiding regulatory and legislative interventions by establishing their own programmes such, as "We Card"	3.24	1.04
66	Promoting a tobacco control focus that is limited to youth issues	3.24	1.26
35	Shifting blame to the victims (e.g. passing youth possession laws to punish youths)	3.24	1.22
Creating illusion of support		**3.27**	**1.09**
54	Using legal and constitutional challenges to undermine federal, state, and local legislative and regulatory initiatives	4.52	0.75
81	Using anti-lobbying legislation to suppress tobacco control advocacy	3.57	1.16
68	Flying in cadre of "experts" to fight local/state legislation	3.43	0.98
39	Creating the illusion of a pro-tobacco grassroots movement through direct mail database and paid-for petition names	3.19	1.21
60	Using international activities to avoid domestic rules on ads, taxation, etc	3.05	1.02
33	Entering false testimony and false data into the public record	2.95	1.20
75	Tying states' MSA money to increases/decreases of smoking prevalence	2.95	1.32
59	Using employees and their families to make campaign contributions that are difficult to track	2.52	1.08
Harassment		**3.26**	**1.19**
43	Intimidating opponents with overwhelming resources	4.38	0.74
32	Using the courts, and threats of legal action to silence opponents	4.19	0.93
37	Harassing tobacco control workers via letters, FOIAs, and legal action.	3.43	1.43
56	Silencing industry insiders	3.19	1.36
23	Hassling prominent tobacco control scientists for their advocacy work	3.00	1.45
3	Infiltrating tobacco prevention and control groups	2.81	1.17
25	Trying to undermine those selling effective cessation products	1.81	1.25
Undermining science		**3.26**	**1.09**
11	Creating doubt about the credibility of science by paying scientists to disseminate pro-tobacco information	3.76	0.77
18	Sowing confusion about the meaning of statistical significance and research methods	3.57	1.12
38	Creating scientific forums to get pro-tobacco information into the scientific literature	3.33	1.24
5	Influencing scientific publication by paying journal editors to write editorials opposing tobacco restrictions	3.10	1.09
71	Creating doubt about the credibility of science by paying scientists to provide expert testimony	3.10	1.22
80	Creating doubt about the credibility of legitimate science by paying scientists to conduct research	3.05	1.16
86	Conducting studies that, by design, cannot achieve a significant result	2.90	1.04
Media manipulation		**2.91**	**1.13**
77	Using advertising dollars to control content of media	3.71	0.96
34	Putting own "spin" on the issues by manufacturing information sources	3.43	1.12
67	Taking advantage of the "balanced reporting" concept to get equal time for junk science	2.86	1.20
69	Ghost writing pro-tobacco articles	2.76	1.22
6	Avoiding the key health questions by saying they are not experts and then not agreeing with the experts	2.71	1.27
84	Misrepresenting facts in situations where there is no time to verify	2.67	0.97
74	Publicly acknowledging the risk of tobacco use, but minimising the magnitude	2.67	1.20
30	Publicising research into "safe cigarettes"	2.48	1.12

Table 1 continued

Number	Statement	Mean	SD
Public relations		**2.85**	**1.10**
12	Using philanthropy to link their public image with positive causes	4.00	0.89
28	Using philanthropy to build a constituency of support among credible groups	3.62	0.80
73	Diverting attention from the health issues by focusing attention on the economic issues	3.48	0.98
51	Distracting attention from the real issues with alternative stances such as accommodation and ventilation	3.38	1.40
88	Asserting that restrictions on tobacco could lead to restrictions on other industries and products	3.38	0.92
14	Minimising importance of misdeeds in the past by claiming they have changed	3.24	1.41
20	Argue that tobacco control policies are anti-business	3.19	1.03
72	Maintaining that the tobacco industry is of critical importance to the economy	3.19	1.08
45	Portraying themselves "responsible", "reasonable" and willing to engage in a "dialogue"	2.90	1.34
78	Misrepresenting legal issues to naive reporters and stock analysts	2.86	1.20
79	Feeding pro-tobacco information to market analysts who are predisposed to accepting and transmitting it	2.86	1.20
15	Representing people as "anti-smoker" instead of anti-smoking	2.81	1.03
82	Developing pro-tobacco media content, such as videos and press releases	2.67	0.97
83	Painting tobacco control activists as extremists	2.67	1.15
55	Pretending that the "real" tobacco control agenda is prohibition	2.57	1.08
57	Casting tobacco control as a civil rights threat	2.52	1.25
49	Portraying tobacco control as a class struggle against poor and minority groups	2.48	0.98
24	Extensive media training for executives who will be in the public eye	2.43	1.12
70	Shifting attention toward lawyers' monetary gains and away from tobacco litigation	2.38	1.20
47	Avoiding losing public debates by overcomplicating simple issues	2.29	1.15
31	Blaming it on "fall-guys" (past or rogue employees) when the industry is caught misbehaving	2.00	1.22
50	Refusing or avoiding media debates where they think they will do poorly	1.71	0.72

ASHRAE, American Society of Heating, Refridgerating and Air-Conditioning Engineers; FOIA, Freedom of Information Act; MSA, Master Settlement Agreement.

entered.[23] The total similarity matrix was obtained by summing across the individual matrices. Thus, any cell in this matrix could take integer values between 0 and 22 (the number of people who sorted the statements); the value indicates the number of people who placed the pair in the same pile. In addition, in this analysis the final matrix was filtered by changing any matrix values of 1 to a 0. In effect, this means that there needed to be at least two participants who place any two statements together in order for them to be considered at all similar. This filtering helps minimise the effects of any errors or spuriousness in sorting on the final results.

The total similarity matrix was analysed using non-metric multidimensional scaling (MDS) analysis[25] with a two dimensional solution. The solution was limited to two dimensions because of ease of use considerations.[26]

The x,y configuration output from MDS was the input for the hierarchical cluster analysis utilising Ward's algorithm[27] as the basis for defining a cluster. Using the MDS configuration as input to the cluster analysis in effect forces the cluster analysis to partition the MDS configuration into non-overlapping clusters in two dimensional space. There is no simple mathematical criterion by which a final number of clusters can be selected. The procedure followed here was to examine an initial cluster solution that was the maximum thought desirable for interpretation in this context. Then, successively lower cluster solutions were examined, with a judgment made at each level about whether the merger seemed substantively reasonable.

The MDS configuration of the statement points was graphed in two dimensions. This "point map" displayed the location of all the brainstormed statements with statements closer to each other generally expected to be more similar in meaning. A "cluster map" was also generated that displayed the original statement points enclosed by polygon shaped boundaries that depict the clusters.

The 1 to 5 importance rating variable was averaged across persons for each item and each cluster. This rating information was first depicted graphically in a "point rating map" showing the original point map with the average rating per item displayed as vertical columns in the third dimension and, second, in a "cluster rating map" that showed the cluster average rating using the third dimension.

Expert panel interpretation of the maps

A panel of 13 tobacco control experts who were members of the larger group was convened for a face-to-face meeting to review and interpret the results of the mapping process. The interpretation session followed a structured process described in detail in Trochim.[21] Participants examined the maps to determine whether they made intuitive sense and to discuss what the maps might imply about the ideas that underlie their conceptualisation. They discussed each cluster until a consensus was reached on an acceptable cluster label. Participants then examined the labelled cluster map to identify any interpretable groups of clusters or "regions". These were discussed and partitions drawn on the map to indicate the different regions. Just as in labelling the clusters, the group then arrived at a consensus label for each of the identified regions. This step-by-step interpretation culminated in a discussion of the overall meaning of the various maps and representations, and in the articulation of a conceptual model of pro-tobacco tactics.

RESULTS

The usual statistic that is reported in MDS analyses to indicate the goodness-of-fit of the two dimensional configuration to the original similarity matrix is called the "stress value". A lower stress value indicates a better fit. In a study of the reliability of concept mapping, Trochim reported that the average stress value across 33 projects was 0.285 with a range from 0.155 to 0.352.[28] The stress value in this analysis was 0.237, which is better (that is, lower) than average.

The pattern of judgments of the suitability of different cluster solutions was examined and resulted in acceptance of an eight cluster solution as the one that both preserved the most detail and yielded substantively interpretable clusters of statements. The 88 statements are shown in table 1 in descending order by average importance within the eight clusters, along with their standard deviations. The point cluster map in fig 2 shows all of the pro-tobacco tactics statements (points) in relation to each other.

Figure 3 shows the cluster rating map where the layers of each cluster depict the average importance rating, with more layers equivalent to higher importance. Note that the average represented by the layers in the map is actually a double

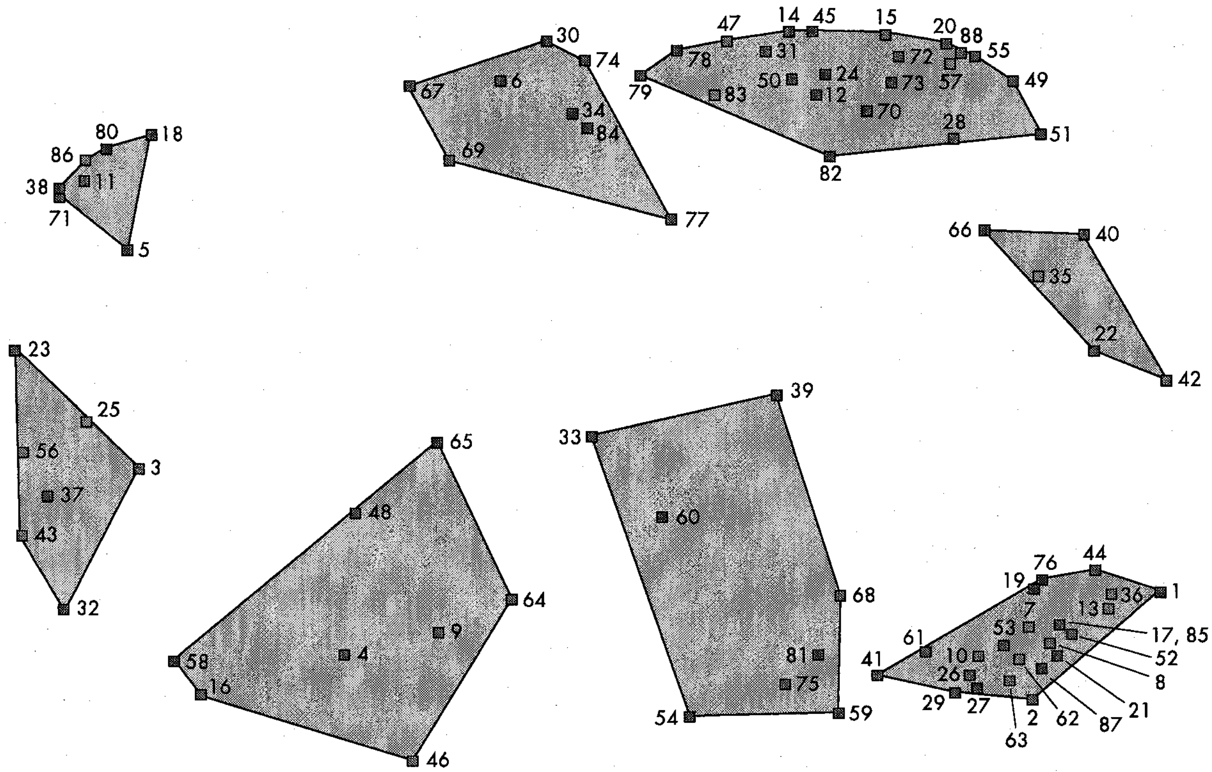


Figure 2 Point cluster map showing the multidimensional scaling arrangement of the 88 statements with the eight cluster solution superimposed.

averaging—across all of the participants and across all of the factors in each cluster. Consequently, even slight differences in averages between clusters are likely to be meaningfully interpretable. The map shows that clusters along the bottom are judged more important in undermining anti-tobacco efforts.

Expert panel interpretation

The expert panel interpreted the map and table in terms of several interesting patterns. The four clusters across the top were thought to describe the messages that the tobacco industry issues or tries to control—what the tobacco industry says. This includes attempts to undermine science and legitimate messages from scientific quarters (Undermining science), the manipulation of the media (Media manipulation), the industry's public relations efforts (Public relations), and the tactics they use to gain control of the public agenda (Usurping the agenda). The four clusters across the bottom describe industry actions—what the tobacco industry does. This includes lobbying efforts (Lobbying and legislative strategy), the use of front groups and artificially created "grassroots" movements (Creating the illusion of support), intimidation (Legal and economic intimidation), and harassment of tobacco control professionals (Harassment).

The participants also interpreted a horizontal dimensionality. Toward the left on the map are clusters that represent tactics that are more hidden or covert in nature. On the right are tactics that tend to be more overt or public in nature. The dimensional interpretation is not meant to suggest that any cluster would be exclusively classifiable into one or the other extreme on a dimension. Undermining science is not exclusively Covert, while Lobbying and legislative strategy is not exclusively public. The relational nature of the map suggests that the clusters vary along the public-covert and message-action dimensions with varying levels of each end point present in each cluster.

Members of the expert panel then suggested that the two dimensions can be viewed as forming four quadrants based on the 2×2 combination of these dimensions and provided a short label for each quadrant: Public + Messages = Issue framing; Public + Action = Lobbying tactics; Covert + Messages = Science PR (public relations); and Covert + Action = Harassment.

Finally, the expert panel discussed these dimensionalities and agreed upon a final labelling for all areas of the map. These features are all depicted in fig 3.

DISCUSSION

The primary purpose of this project was the development of a conceptual framework that describes the tactics the tobacco industry uses to undermine tobacco control programmes. Such a framework may be used in a variety of ways. Here, we discuss the potential utility of the framework for evaluation measurement development, strategic planning, and to support efforts to classify and analyse tobacco industry documents.

Use in measurement development

Figure 3 could be used as the basis for the development of an index of tobacco tactics. To do so would require that each of the clusters be operationalised. The statements within each cluster suggest potential elements that might be measured as part of the index. For instance, one statement in the cluster Lobbying and legislative strategy was "Promoting smokers' rights legislation". This could be operationalised at the state level as the number of proposed bills or a measure of the amount of relevant legislative committee activity. Another statement was "Lobby to assure that funds are diverted to ineffective tobacco control activities". Here, measures of tobacco control programme funding and evidence of lobbying activities might be utilised. In this manner, the statements in each cluster can act as prompts or suggestions for potential operationalisations.

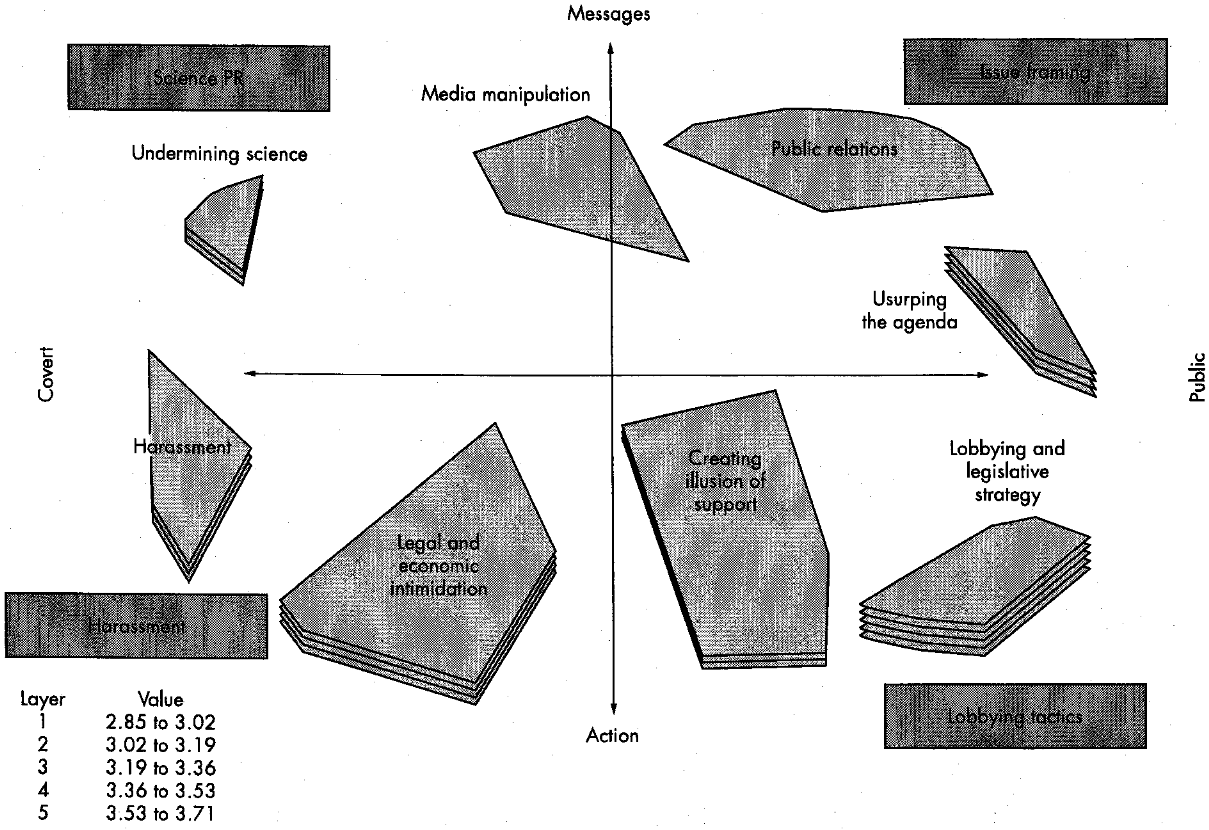


Figure 3 Concept map showing clusters, cluster labels, relative importance ratings, and expert's interpretations of dimensions and regions.

In addition, the overall structure of the map suggests how such an index might be aggregated. For instance, sub-index scores for the clusters Public relations and Usurping the agenda can be aggregated into a total score that represents Issue framing. Moving one level up the hierarchy, the four sub-index scores that represent the quadrants can be aggregated into an overall index of Pro-tobacco tactics.

We know from the map results that the expert panel did not view all of the tactics as equally important. This importance rating information can be incorporated into the development of an index such that sub-index scores for each cluster are weighted by the average importance and the final index aggregation weighted by quadrant importance averages.

Use in tobacco control planning

The conceptual map can provide a high level strategic view of industry tactics that can help tobacco control planners better anticipate the tactics that the industry might use in certain circumstances. For instance, a potentially useful aspect of the map that surfaced in the interpretation can be seen as one moves from the right to the left side. The overt public industry tactics on the right of the map tend to be ongoing activities that the industry does routinely. Like virtually all other major industries, the tobacco industry has ongoing public relations and lobbying efforts as suggested in the clusters on the right.

But, how does the industry change its tactics in response to the perceived threat of increasing tobacco control efforts? The map and the expert panel suggested that they probably do so by moving from upper right toward lower left. Initially they most likely augment their public relations and lobbying efforts. If the tobacco control efforts become salient enough, the map suggests that the industry will be pressured increasingly to the more covert activities on the left side that include undermining science, legal and economic intimidation, and harassment.

The map thus provides a high level strategic model of the industry's response to increased tobacco control efforts. This model can be used in tobacco control planning to better anticipate what the industry may do next.

Use in tobacco document analysis

The conceptual framework can be used for classifying industry documents specifically with respect to industry tactics and, as such, would augment and extend existing document classification and indexing procedures like the UCSF/ANRF Tobacco Documents Thesaurus. For example, each document could be classified for its relevance to the eight cluster areas. Once done, it would be immediately possible to retrieve all documents that provide evidence for a particular type of tactic (for example, cluster), or display all documents that reflect a broader cross-cutting (for example, a column category like "covert") activity on the part of the industry.

For example, consider the cluster Creating illusion of support in the lower part of the map. The statements in that cluster (table 1) indicate several key sub-topics that are relevant and could help guide both the searching and classifying of documents.

For instance, for the statement "Creating the illusion of a pro-tobacco grassroots movement through direct mail database and paid-for petition names", one document identified as relevant is a 1994 Philip Morris presentation that described their efforts to create the illusion of support: "We also are mobilising support among our consumers. Consumers who respond to our brand promotions receive an insert with their fulfillment packages . . .so far, more than 400,000 consumers have responded, and the programme has generated some 80,000 letters to Capital Hill, about 10,000 per month."[29] Similarly, for the statement "Using employees and their families to make campaign contributions that are difficult to track" one relevant document is a 1997 Brown and Williamson letter which states that "as a Brown & Williamson employee, you can play a major role in influencing elections, the future of our business and, of course, our respective jobs" by "making contributions to the B&W Employee Political Action Committee". The letter discusses previous contribution levels for 1996 and options for method of contributing (payroll deductions or personal checks) and asks for a $200 contribution from each eligible participant.[30] Or, for the statement "Flying in cadre of 'experts' to fight local/state legislation", a 1993 Philip Morris document describes the objective "to support the defeat of unwarranted smoking restrictions and to discourage unfair discrimination against smokers". Goals and tactics were: "promotion of ETS in the context of indoor air quality and use of experts to directly and indirectly influence legislation, rulemaking and standards in relation to ETS and workplace smoking issues."[31] These examples are meant to illustrate how the conceptual map can be used both as a suggestive device when searching the documents and as an expert derived hierarchical thematic taxonomy of pro-tobacco tactics that can be useful in coding and organising the documents subsequently identified.

Another document related application would be to develop a cross referencing between the map categories and other classification systems such as the UCSF/ANRF Tobacco Documents Thesaurus. For example, the Thesaurus includes the terms "lobbying", "industry front group", and "industry sponsored research" which could be linked with the map categories Lobbying and legislative strategy, Creating illusion of support, and Undermining science, respectively. This type of cross referencing would enable the tobacco documents to be accessed immediately through different conceptual schema that were devised for different purposes, without having to reclassify all documents from scratch.

In addition to its use in addressing the three issues described above, the conceptual framework can act as an organising device that encourages greater synergy between the three activities. For example, if in a local context, tobacco control planners determine that the industry is likely to increase its efforts in creating the illusion of support in the immediate future, the planners could examine that cluster on the map to help determine the specific tactics the industry might use, to think about how to measure or track the industry's effort in this area, and to access the tobacco document evidence relevant to that cluster that describes the history of similar activities in other contexts.

Additional work could enhance the utility of this framework for document analysis. In this study, participants were asked to brainstorm industry tactics from their point of view and in their own language. This creates, in effect, a map that is decidedly anti-tobacco in its perspective. But the tobacco documents themselves are generated from an opposing perspective, using euphemisms and industry code terms designed to portray their pro-tobacco efforts in a good public light. Where anti-tobacco researchers might, for instance, talk about the industry "paying scientists to conduct research to create doubt about legitimate science" (statement 80), it is unlikely that industry documents would describe their activities in a similar manner. Document searches that rely directly on the language of the map are unlikely to be fruitful or get at the desired topics. This suggests that it would be useful to develop the type of cross referencing to the Thesaurus that was discussed above.

Finally, there were activities of the tobacco industry, such as manipulating product chemistry or price, that were not included in this map because the focus in this project was on specific activities/tactics the industry uses to undermine tobacco control programmes. The manipulation of chemistry or price were not perceived by participants as "tactics" for undermining tobacco control per se. Despite not being considered industry tactics for undermining tobacco control programmes, the importance of these issues is undeniable and they need to be addressed in comprehensive evaluations of tobacco control programmes.

Regardless of the real world potential uses for the conceptual map, the structure is an intriguing one in its own right. It summarises a very complex area concisely and provides a compelling theoretical model that needs to be tested and extended empirically in follow up work. Replications of this study could be used to determine the reliability and generalisability of the model. In addition, the model is general enough at its highest level to be a potential framework that might be applied to understanding the tactics of other industries that attempt to undermine the legitimate work of public health programmes.

What this paper adds

A major challenge in evaluating tobacco control efforts is the need to measure tobacco industry counter-efforts and their effects. Currently, no overarching conceptual model exists to guide operationalisation of measures of industry tactics that might be useful for evaluation. This study used a web based multivariate concept mapping methodology with a panel of tobacco control experts to develop a conceptual model of the tobacco industry interference with tobacco control programmes.

The resulting conceptual maps summarise the tactics used by the industry and their relationships to each other, and suggest a possible hierarchy for measures that can be used in statistical modelling of industry tactics and for review of industry documents. Finally, the maps enable hypothesis of a likely progression of industry reactions as public health programmes become more successful, and therefore more threatening to industry profits.

ACKNOWLEDGEMENTS

This article was supported by contract number N01-CP-95030 from the National Cancer Institute. Its contents are solely the responsibility of the authors and do not necessarily represent the official view of the National Cancer Institute.

....

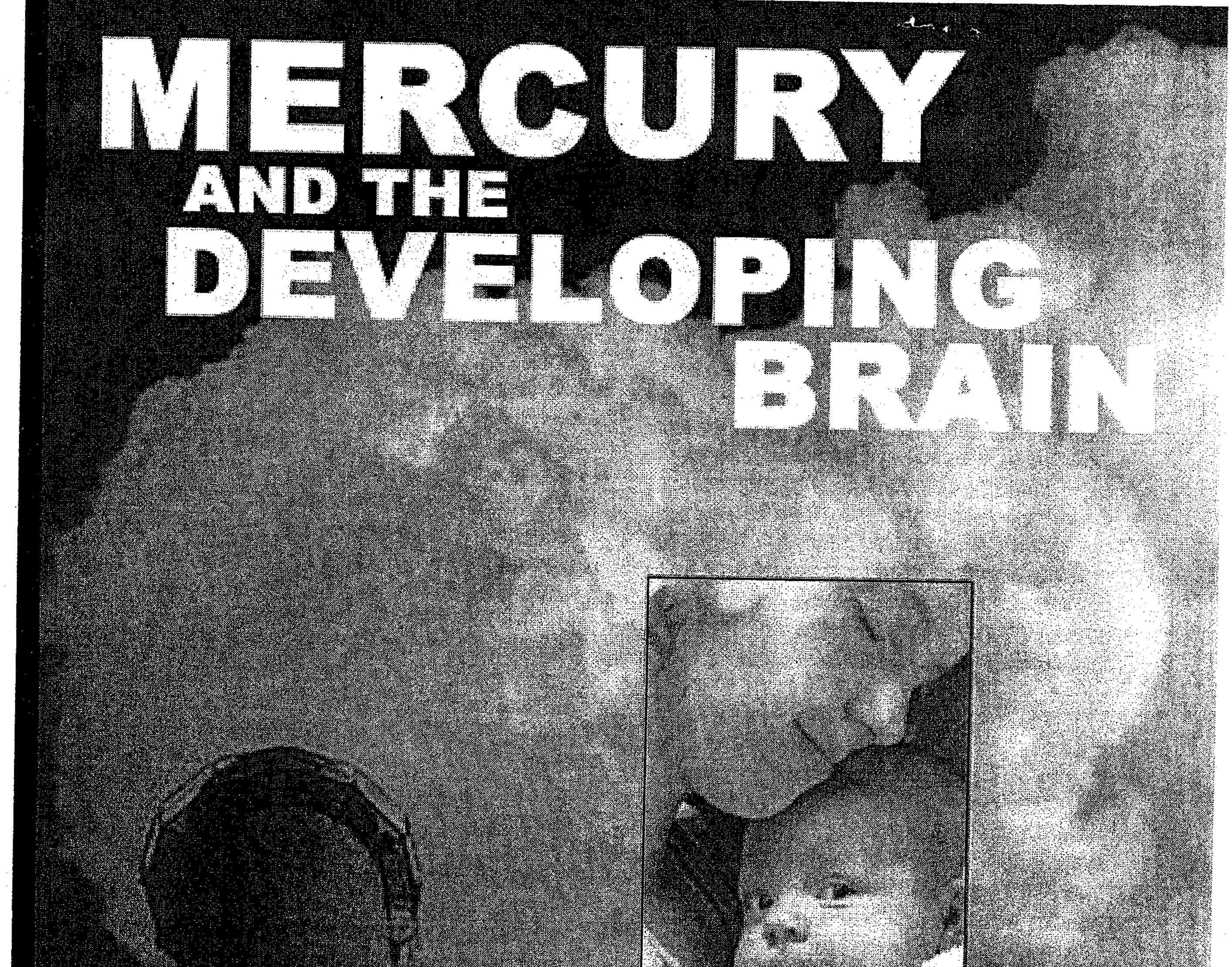
Authors' affiliations

W M K Trochim, Department of Policy Analysis & Management, Cornell University, Ithaca, New York, USA
F A Stillman, Institute of Global Tobacco Control, Johns Hopkins University, Baltimore, Maryland, USA
P I Clark, C L Schmitt, Battelle Memorial Institute, Centers for Public Health Research and Evaluation, Baltimore, Maryland, USA

REFERENCES

1 **Saloojee Y**, Dagli E. Tobacco industry tactics for resisting public policy on health. *Bull World Health Organ* 2000;**78**:902–10.
2 **Glantz SA**, Barnes DE, Bero L, *et al*. Looking through a keyhole at the tobacco industry. *JAMA* 1995;**274**:219–24.
3 **Cummings KM**, Sciandra R, Gingrass A, *et al*. What scientists funded by the tobacco industry believe about the hazards of cigarette smoking. *Am J Public Health* 1991;**81**:894–6.
4 **Sweda ELJ**, Daynard RA. Tobacco industry tactics. *Br Med Bull* 1996;**52**:183–92.
5 **Zeltner T**, Kessler D, Martiny A, *et al*. *Tobacco company strategies to undermine tobacco control activities at the World Health Organization.* Geneva: World Health Organization, 2000. URL: http://filestore.who.int~who/home/tobacco/tobacco.pdf
6 **Samuels B**, Glantz SA. The politics of local tobacco control. *JAMA* 1991;**266**:2110–7.
7 **Goldstein AO**, Bearman NS. State tobacco lobbyists and organizations in the United States: crossed lines. *Am J Public Health* 1996;**86**:1137–42.
8 **Jacobson PD**, Wasserman J. The implementation and enforcement of tobacco control laws: policy implications for activists and the industry. *Journal of Health Politics, Policy & Law* 1999;**24**:567–98.
9 **Givel MS**, Glantz SA. Tobacco lobby political influence on US state legislatures in the 1990s. *Tobacco Control* 2001;**10**:124–34.
10 **Traynor MP**, Begay ME, Glantz SA. New tobacco industry strategy to prevent local tobacco control. *JAMA* 1993;**270**:479–86.
11 **Aguinaga S**, Glantz S. The use of public records acts to interfere with tobacco control. *Tobacco Control* 1995;**4**:222–30.

12 **Bialous SA**, Fox BJ, Glantz SA. Tobacco industry allegations of "illegal lobbying" and state tobacco control. *Am J Public Health* 2001;**91**:62–7.
13 **Author unknown**. Synar/ASSIST Task Force. Philip Morris. 1993. Bates No. 2023961347-1359 Accessed 1 November 2001. www.pmdocs.com <http://www.pmdocs.com> Merlo E. Vendor Conference Draft. Philip Morris Corporate Affairs. December, 1993. Draft speech. Bates No. 2040863440-3463.
14 **Author unknown**. (ASSIST program and Synar Amendment to ADAMHA). July 1992. Philip Morris. Bates No. 2048621152-1175. Accessed 5 November 2001. URL: www.pmdocs.com
15 **Manley M**, Lynn W, Epps R, *et al*. The American Stop Smoking Intervention Study for cancer prevention: an overview. *Tobacco Control* 1998;**6**(suppl 2):S5–11.
16 **Tisch AH**. Lorillard article for NY Assoc. of Tobacco and Candy Distributors. Lorillard Tobacco Company.(1992) Bates No. 92761408-9. Accessed 5 August 2001. URL: http://www.lorillarddocs.com
17 **Stillman F**, Hartman A, Graubard B, *et al*. The American Stop Smoking Intervention Study: conceptual framework and evaluation design. *Evaluation Review* 1999;**23**:259–80.
18 **University of California San Francisco**. URL: http://www.library.ucsf.edu/tobacco/thesaurus.html
19 **Malone RE**, Balbach ED. Tobacco industry documents: treasure trove or quagmire? *Tobacco Control* 2000;**9**:334–8.
20 **Glantz S**. The truth about big tobacco in its own words. *BMJ* 2000;**321**:313–4.
21 **Trochim W**. An introduction to concept mapping for planning and evaluation. *Evaluation Program Planning* 1989;**12**:1–16.
22 **Rosenberg S**, Kim MP. The method of sorting as a data gathering procedure in multivariate research. *Multivariate Behavioral Research* 1975;**10**:489–502.
23 **Weller SC**, Romney AK. *Systematic data collection*. Newbury Park, California: Sage Publications, 1988.
24 **Coxon APM**. Sorting data: collection and analysis. Sage University Papers on Quantitative Applications in the Social Sciences, 07-127. Thousand Oaks, California: Sage Publications, 1999.
25 **Davison ML**. *Multidimensional scaling*. New York: John Wiley and Sons, 1983.
26 **Kruskal JB**, Wish M. *Multidimensional scaling*. Beverly Hills, California: Sage Publications, 1978.
27 **Everitt B**. *Cluster analysis*, 2nd ed. New York: Halsted Press, a division of John Wiley and Sons, 1980.
28 **Trochim W**. Reliability of concept mapping. Paper presented at the Annual Conference of the American Evaluation Association, Dallas, Texas, November 1993. Accessed 15 August 2001. URL: http://trochim.human.cornell.edu/research/reliable/reliable.htm
29 **Philip Morris**. Final draft: EM presentation 30 March 1994. Philip Morris. Bates No. 2024007084-7109. Accessed 21 August 2001. URL: http://www.pmdocs.com/
30 **Brown & Williamson**. [Memo: Dear Fellow B&W Employee] Brown & Williamson, 13 February 1997. Bates No. 621960913. Accessed 15 August 2001. URL: http://www.bw.aalatg.com/public.asp
31 **Philip Morris**. Public Smoking. Philip Morris, 1993. Bates No. 2024234063–4075. Accessed 21 August 2001. URL: http://www.pmdocs.com


MERCURY
AND THE
DEVELOPING
BRAIN
CLEAR
THE
AIR

Mercury and the Developing Brain

Introduction

Children are most vulnerable to mercury exposure, whether exposed in utero or as young children. Mercury affects the developing brain, causing neurological problems that manifest themselves as vision and hearing difficulties, delays in the development of motor skills and language acquisition, and later, lowered IQ points, problems with memory and attention deficits. These developmental deficits may translate into a wide range of learning difficulties once children are in school.

This report explains the sources of mercury in the environment and how people are exposed. The physical changes that occur in the developing brain due to mercury exposure during pregnancy are described along with how these changes later translate into learning difficulties in school. The report estimates the societal and economic impacts of mercury exposure in terms of the cost of special education in the U.S. and the societal benefits of reducing mercury emissions.

The information in this report is timely. Numerous policy options are being considered by state, federal and international lawmakers to reduce mercury emissions to the environment. Stringent regulations, implemented as quickly as possible, must be enacted to help reduce the level of mercury exposure to children.

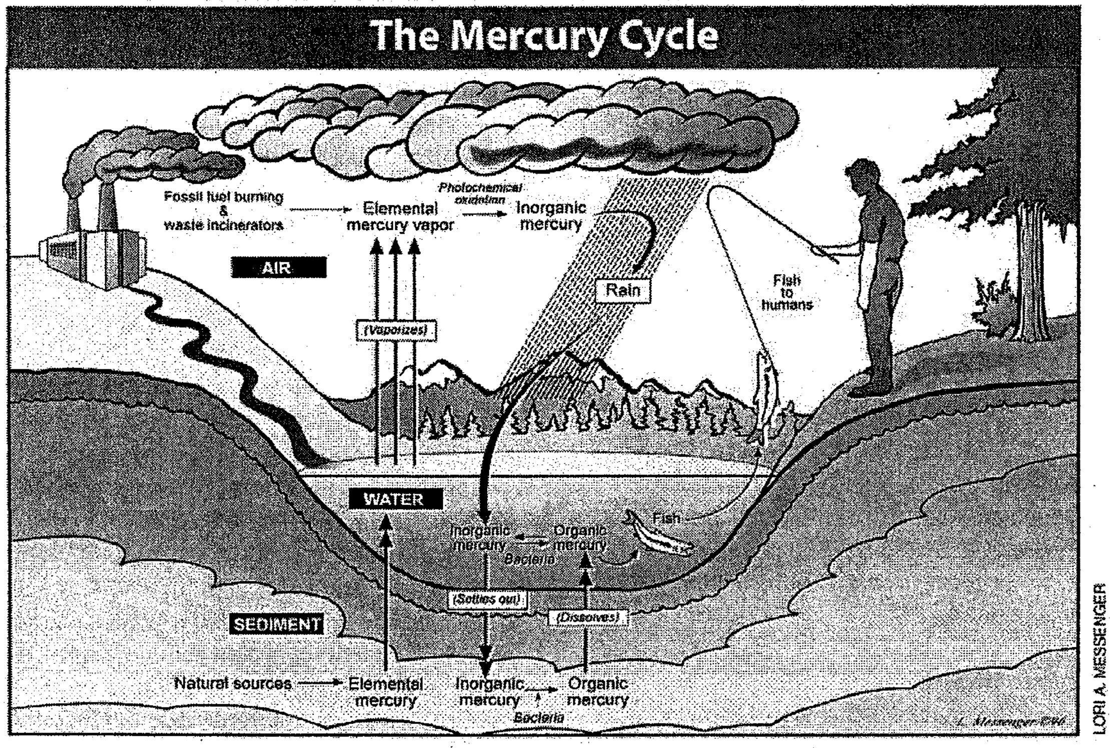
Methylmercury Exposure in the U.S.

Methylmercury is a neurotoxin – a substance that damages, destroys, or impairs the functioning of nerve tissue. In the U.S., the general population is exposed to various forms of mercury through inhalation, consumption of contaminated food or water, and exposure to substances containing mercury, such as vaccines. Different chemical types of mercury can adversely affect several organ systems, with the severity of effects depending largely on the magnitude and timing of the exposure (i.e., during fetal development or as a child or adult).[1] Outside of occupational settings, methylmercury is the most toxic form of mercury to which humans are regularly exposed and this form of mercury is the focus of the health impacts discussed in this report. Exposure to methylmercury in the U.S. is almost exclusively from eating fish and shellfish.

Methylmercury poses the greatest hazard to the developing fetus. It passes easily through the placenta and impairs the development of the brain and nervous system. Prenatal methylmercury exposure from maternal consumption of fish can cause later neurodevelopmental effects in children.[2] Infants appear normal during the first few months of life, but later display subtle effects. These effects include poor performance on neurobehavioral tests, particularly on tests of attention, fine motor function, language, visual-spatial abilities (e.g., drawing) and memory. These children will likely have to struggle to keep up in school and might require remedial classes or special education.[3]

Methylmercury exposure prior to pregnancy is as critical as exposure during pregnancy because methylmercury stays in the body for

Figure 1. The Mercury Cycle

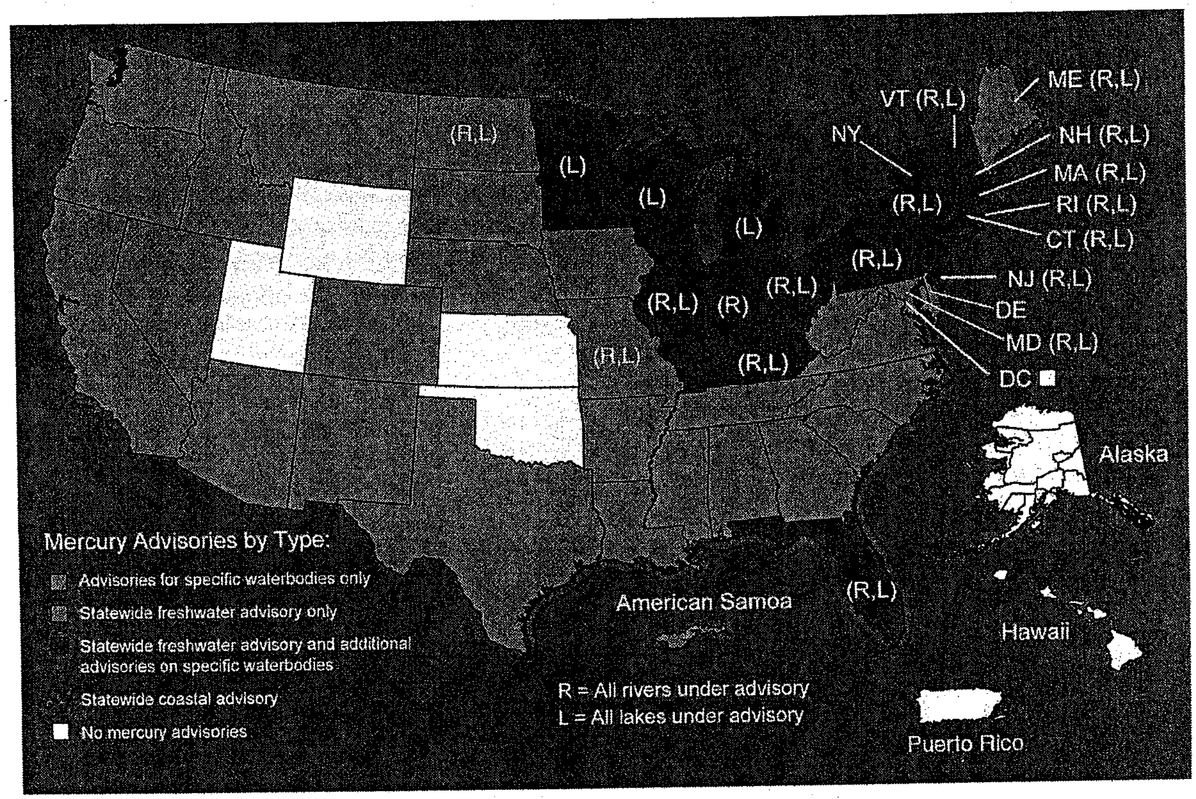


Elevated levels of methylmercury in fish have prompted concerns about the public health hazards from methylmercury exposure. Despite the known nutritional and health benefits from eating fish, in 2003, public health agencies in 45 states issued fish consumption advisories warning citizens to limit how often they eat certain types of fish because the fish are contaminated with high levels of mercury.[7] Twenty-one states have issued mercury advisories for fish in *every inland lake or river.*[8] (Figure 2.)

The U.S. Food and Drug Administration and EPA specifically warn pregnant women, women of childbearing age, nursing mothers, and young children not to eat shark, swordfish, king mackerel, or tilefish. The advisory warns the same populations to limit their consumption of albacore "white tuna" or tuna steaks to six ounces or less per week and fish that have lower levels of mercury, such as shrimp, canned light tuna, salmon, pollock, and catfish, to 12 ounces or less per week. (Six ounces of fish is an average cooked meal, about the size of a can of tuna.[9])

In 2004, EPA indicated that 1 in 6 women of childbearing age has mercury levels in her blood above EPA's current health threshold.[10] Nationally, this means that as many as 630,000 of the four million babies born each year – or 15 out of every 100 babies - are at risk of developmental problems due to mercury exposure in utero.[11]

Figure 2. Mercury Fish Consumption Advisories



Methylmercury and Brain Development

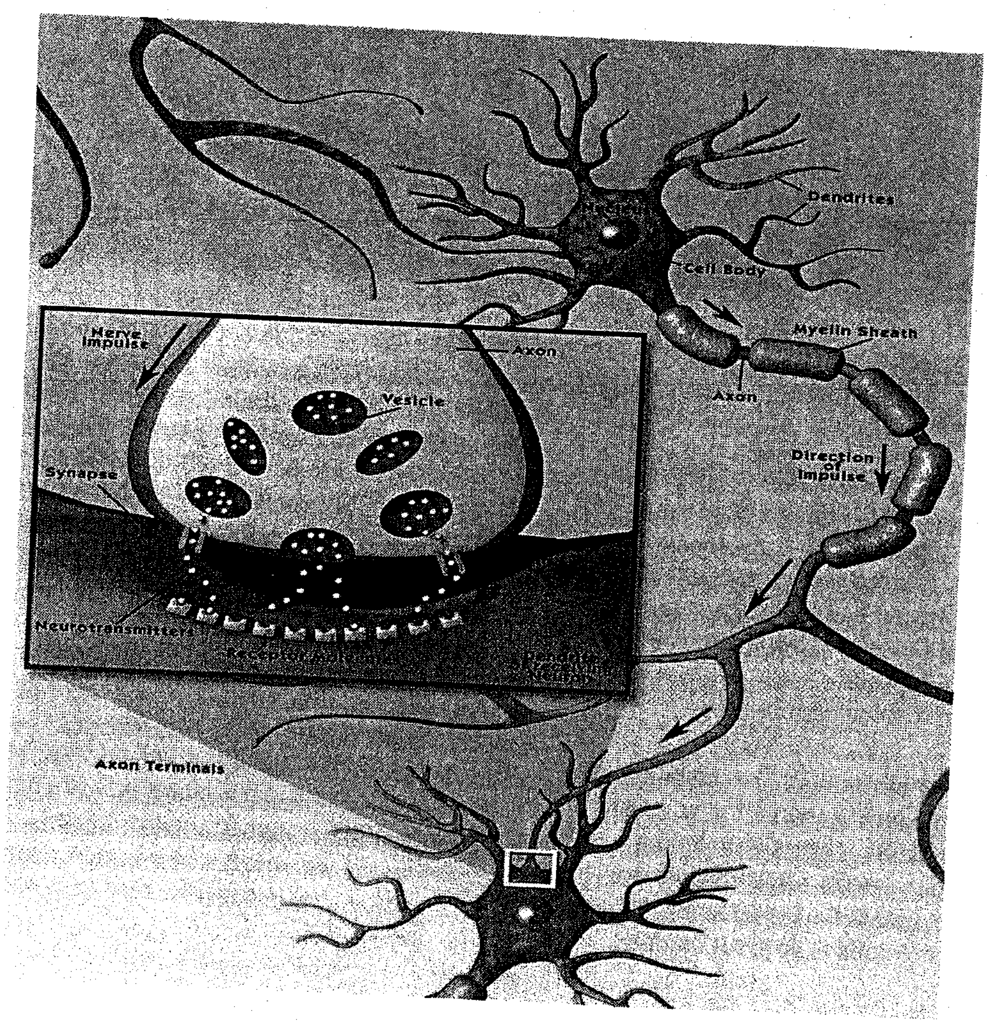
Understanding how the brain forms is essential to our understanding of how methylmercury affects brain development. The detrimental effects of methylmercury exposure that become evident in a young child are thought to result from changes that occur during the formation and organization of brain cells in the developing embryo.

The fully formed brain, with its elaborate and precisely balanced network of at least 100 billion nerve cells, begins developing a mere three to four weeks after conception. During the nine months of pregnancy, the production of nerve cells is so great that at times as many as 50,000 cells are created each second to provide the necessary number for the adult brain. Brain development is so complex that at least half of our genetic code is devoted to producing this organ. While genetics largely determine brain development, environmental factors are also involved from almost the beginning of embryonic life. These environmental factors include the health and nutritional status of the mother and her contact with potentially toxic substances like alcohol, tobacco and other drugs. Methylmercury exposure at this critical time of fetal growth is also an environmental factor that can have a detrimental effect on brain development.

Neurons Wire the Brain

The building block of the brain and central nervous system is the nerve cell, or neuron. A neuron is a specialized cell that transmits information to other nerve cells, muscles or glands. (Figure 3.) Neurons consist of a cell body containing the nucleus and an axon, an electricity-conducting fiber that conducts the nerve signal away from the cell body. The axon gives rise to many smaller axon branches before ending at nerve terminals. Another extension of the cell body includes dendrites, which extend from the neuron cell body like branches of a tree and receive the incoming signal from other neurons. Their function is to conduct a nerve impulse toward the cell body. Synapses are the actual gaps between neurons through which nerve impulses travel, like the gap in spark plugs. Hollow tubes called microtubules run the length of the neuron and function not only as the "skeleton" or supporting structure for the cell but act as conveyor belts for transporting cellular components within the cell.

Figure 3. The Healthy Neuron



How Neurons Work

Neurons signal by transmitting electrical impulses along their axons. A layer of specialized fatty cells produce the myelin sheath, which insulates the axon and helps speed the transmission of electrical impulses. The electrical impulses are transmitted at speeds up to several hundred miles per hour. The impulses are formed by a complex interaction of electrically charged sodium and potassium atoms, which move in and out of the neuron through the cell membrane, creating a tiny voltage change. At the end of the axon, the voltage change causes the release of calcium atoms that in turn signal the release of specific chemical messengers, called neurotransmitters. The neurotransmitters cross the synapse and bind to receptors on the dendrite of the target neuron, starting an electrical impulse in it. Thus, the impulses are transmitted from neuron to neuron in a domino effect – faster than a lightening strike.

Neurons and Development of the Central Nervous System

Neurons are initially produced in the neural tube – the primitive brain that will form the hindbrain, midbrain and forebrain. The neurons produced in the neural tube migrate in a precise and complex sequence to a final destination in the brain which is determined both by genetics and by the activity of various proteins that influence the type of neuron that will be formed. Neuron migration does not occur at a constant rate throughout development, but occurs in "waves" depending on cell type. The neurons form each of the brain's specific structures and grow long distances to find and connect, through the formation of synapses, to other specialized neurons. Although neurons retain the ability to make new synapses throughout life, the developmental period is critical for the basic formation of the intricate and specific circuits of the nervous system. In addition, throughout all stages of development the number of neurons is reduced through a process known as apoptosis, or programmed cell death. This process is necessary as only about one-half of the neurons generated during development are needed in the adult brain.

All neurons in the central nervous system pass through this same sequence of events. However, neurons in different regions of the brain mature at different times. Each region of the brain has an individual timetable of development that is fixed and cannot be delayed. Periods of intense neuron proliferation have been shown in most brain regions in humans. The timing of such growth spurts can occur over a short period of time, frequently over a period of only a few hours. The migration of neurons to their correct destination in the brain must occur in proper sequence and at the proper time if normal brain development is to result. Should migration of any subset of neurons be delayed or interfered with, subsequent neurons will either be blocked or will pass the delayed neurons and lead to displacement of these cells in the brain. Normal function requires that certain cells with certain characteristics are located in the correct location. Different learning or behavorial effects may result from exposure to the same agent at different times in brain development, depending on the location in the brain where susceptible neurodevelopmental events are taking place at the time of the exposure.

How Methylmercury Affects the Brain

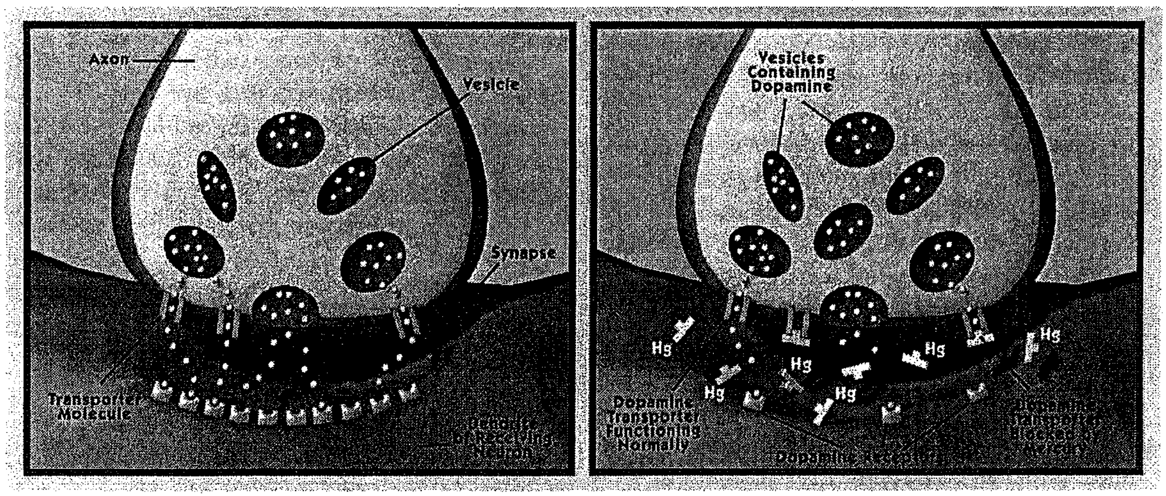
Numerous studies have confirmed that methylmercury exposure changes the function and structure of the central nervous system. In adults, the damage is typically localized in specific areas of the brain, but when exposure to methylmercury occurs in utero or at an early age, the damage to the central nervous system is generalized and widespread. There are several ways by which methylmercury causes developmental damage to the central nervous system which makes the fetus and the young brain far more vulnerable than the adult brain. The mechanisms by which methylmercury affects neurodevelopment are listed below.

- **Methylmercury decreases the transmission of impulses across the synapse.**

Methylmercury decreases the electrical activity of neurons by interfering with the transfer of sodium across the cell membrane. It also blocks the release of calcium ions, thus preventing the release of neurotransmitters such as dopamine. (Figure 4.) Dopamine is a critical neurotransmitter that is necessary for a wide variety of functions such as attention, thinking and motor skills.

Parkinson's disease, mood disorders and deficits in attention, motor control and perception have all been linked to dysfunction of the dopamine system. Research in animals has shown that prenatal exposure to methylmercury resulted in loss of motor control when the exposure occurred during the critical time period when dopamine neurons were developing into mature cells. The dose of mercury in this animal study was comparable to the amount of methylmercury found in the brains of infants from fish-eating populations.

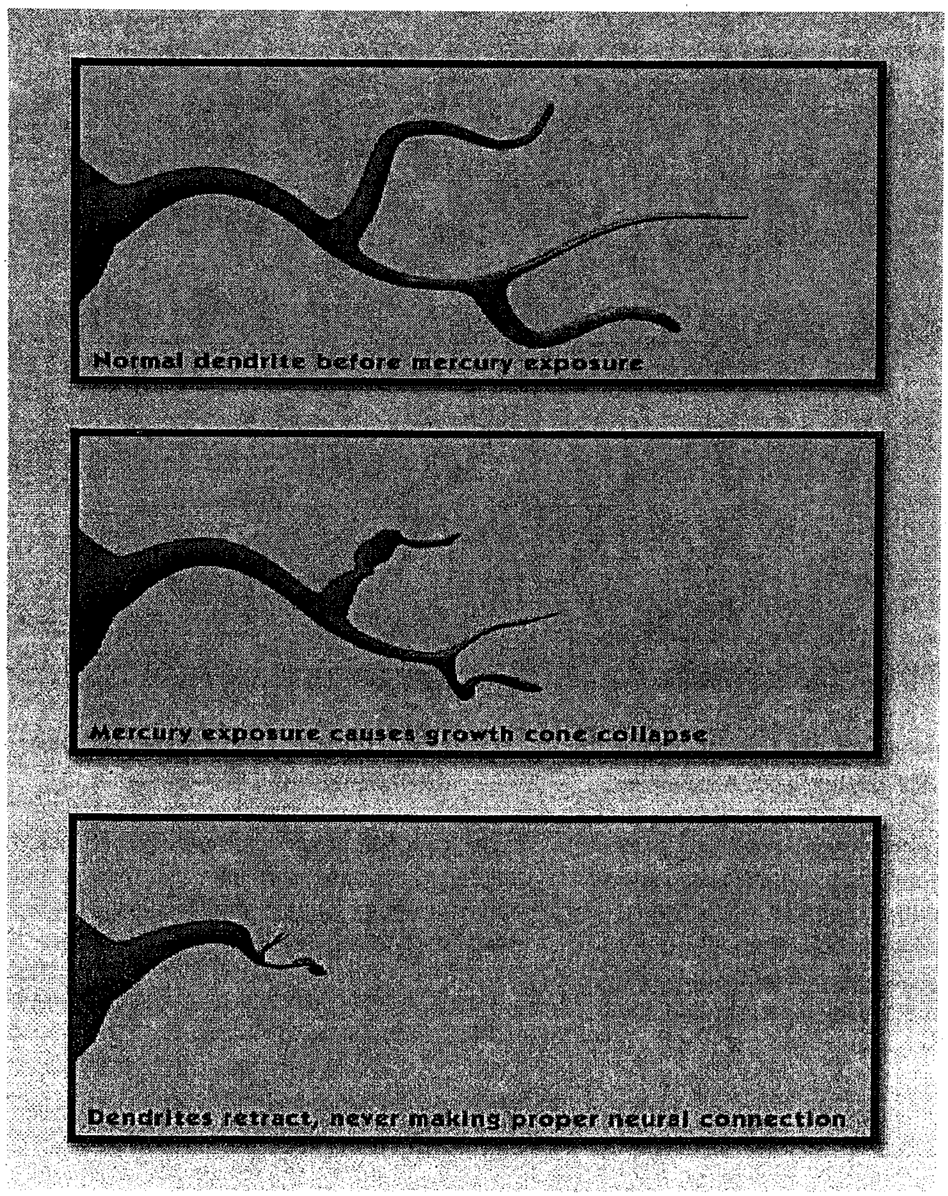
Figure 4. Methylmercury blocks the release of neurotransmitters such as dopamine.



Methylmercury hinders the formation of axons and dendrites.

To complete the circuit of the central nervous system, axons must stretch out to their final destination to meet the dendrites of their connecting neurons. Growth cones - special cells at the tip of the axon - receive chemical signals that tell the growth cone whether to move forward, stop or change direction. Laboratory studies with immature neurons have demonstrated that methylmercury prevents the growth of axons and dendrites by causing the growth cones and the nerve extensions to retract, thus never making the proper connections. (Figure 5.)

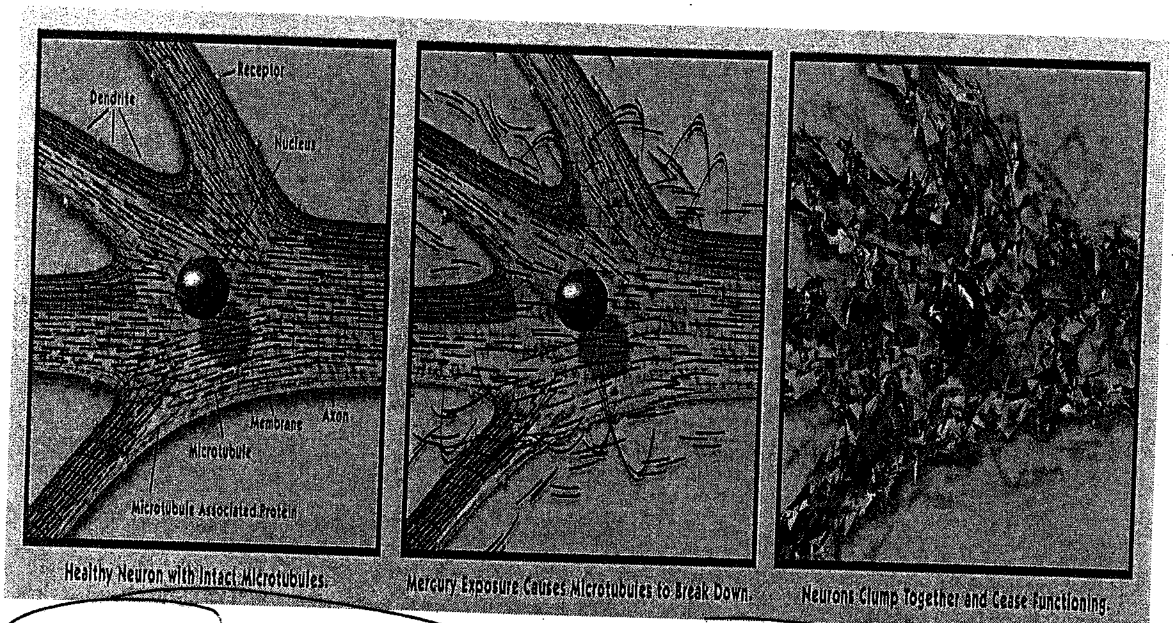
Figure 5. Methylmercury prevents the growth of axons and dendrites.



Methylmercury targets the cell structure and disturbs neuron migration.

A very important feature of prenatal methylmercury exposure is the affect methylmercury has on the cell microtubules. These structures not only provide the structural support for the cell, they play a critical role in cell production, neuron migration and the extension and stabilization of axons. Exposure to methylmercury is known to cause microtubules to break down and leave the neuron stripped of its protective membrane. These neurons clump together forming aggregates that cannot function normally. The result is abnormal arrangement of neurons in the brain. Interference with microtubules causes brain lesions that are consistent with those found in infants exposed in utero to methylmercury during the early stages of pregnancy.

Figure 6. Methylmercury targets the cell structure and disturbs neuron migration.



Healthy Neuron with Intact Microtubules. Mercury Exposure Causes Microtubules to Break Down. Neurons Clump Together and Cease Functioning.

Methylmercury stops cell division and the formation of new neurons.

Prenatal methylmercury exposure is associated with reduced brain size and weight and a reduced number of neurons. Methylmercury impairs cell production by stopping the process of cell division and interfering with the genes that regulate the cell growth cycle.

Methylmercury induces apoptosis (programmed cell death).

While apoptosis is a necessary process during the development of the brain, methylmercury induces this process with the result that the cells die at the wrong time.

Effects of Methylmercury Exposure in Schoolchildren

The most severe effects of methylmercury exposure were reported after high-dose poisoning episodes in Japan beginning in the 1950s and in Iraq in the 1970s. Children exposed to methylmercury in utero suffered severe adverse effects including mental retardation, cerebral palsy, deafness, blindness and dysarthria (a speech disorder that is due to a weakness or lack of coordination of the speech muscles). Sensory and motor impairment were also documented in adults. While poisoning episodes like those in Iraq and Japan are rare, chronic low-dose methylmercury exposure from maternal consumption of fish is common and has been associated with more subtle neurodevelopmental effects in children.

In the 1990s, methylmercury effects were studied in newborns and children in three large investigations in the Faroe Islands, the Seychelles Islands and New Zealand, as well as in other smaller studies in French Guiana and the Amazon.[12] The Seychelles Islands study consisted of 779 mother-infant pairs where the mothers consumed 12 meals per week of fish with low methylmercury levels. Neurodevelopmental tests were performed on the children at approximately 6 months, 1½ years, 2½ years, 5½ years, and 9 years. No effects were observed. The Faroe Islands study included about 900 mother-infant pairs where the mothers consumed 1-3 meals per week of fish with low average methylmercury levels and 1 meal a month of pilot whale meat containing high average methylmercury levels. Neurodevelopmental tests were conducted at 7 years of age. Dysfunctions in language, attention, and memory were observed in the Faroes children.

In the New Zealand study, 38 children of mothers with high mercury levels were paired with children from mothers with low mercury levels. At 6 years of age, 237 children were assessed, using tests similar to those used in the Seychelles study. Similar to the Faroe Islands study, the New Zealand children appeared entirely normal during the first few months of life, but later displayed adverse effects as documented by a battery of neurodevelopmental tests.

Scientists are not sure why the results of the studies differ. Maternal exposure to methylmercury in the studies was comparable and is unlikely to account for the different findings.[13] Among the other differences in the studies that may account for the different findings are differential genetic susceptibility of the population and different patterns of methylmercury exposure (continuous v. episodic).[14] In considering these differences however, the National Research Council advised the U.S. EPA, as a matter of prudent health practice, to use the results of the Faroe Islands and other positive studies for assessing the public health risk of mercury exposure in the U.S.

The tests administered in the Faroe Islands study were neuropsychological tests designed to assess specific brain functions (e.g., attention, motor performance and memory). By comparison, tests administered in the New Zealand study yielded scores that represented the performance of the brain over a number of domains (e.g., IQ). All of the tests assessed functions that are important for a child's ability to learn, remember and succeed in school. The Faroe Islands and New Zealand studies showed a significant correlation between impairment in the areas of language, attention and memory, and prenatal methylmercury exposure.[15] Table 1 describes the tests given in the Faroe Islands and New Zealand and how poor scores on these tests are relevant to school performance.

In 2000, the National Research Council of the National Academies of Sciences reviewed all of the available scientific data on human exposure to methylmercury. It concluded that neurobehavorial deficits of the magnitude reported in the Faroe Islands and New Zealand (as measured by the test results) are likely to be associated with increases in the number of children who have to struggle to keep up in a normal classroom or who may require remedial classes or special education.[16] Data from the Faroe Islands also indicates that adverse health effects associated with methylmercury exposure may not be reversible, increasing the significance of these effects.[17]

Table I. Tests Administered to Children to Assess the Effects of Prenatal Methylmercury Exposure

Mercury Study	Test Administered	Function Assessed by Test	Test Relevance to School Performance
Faroe Islands	Finger Tapping (Oscillation) Test	This procedure measures motor speed and performance. By examining performance on both sides of the body, inferences may be drawn regarding possible disorder of the motor nerves.	Difficulty with fine motor skills such as writing.
	Continuous Performance Test Reaction Time	Vigilance, attention, information processing speed. Identifies children with potential learning disabilities.	Intelligence, school behavior and performance especially in the areas of sustained attention and distractibility.
	Bender Copying Errors	Diagnoses the maturity of visual-motor perception in young children. Identifies problems with visual motor perceptual functioning, such as ability to process and interpret visual information about where objects are in space.	Math performance, classroom performance (e.g., shifting gaze between objects at a distance(writing on the board), to close-up objects (a book at the desk).
	Boston Naming Test	Expressive vocabulary. Specifically assesses confrontational naming skills – child has to identify line drawings of common objects under time pressure. Cues are given if child cannot respond spontaneously.	Reading, school performance.
	California Verbal Learning Test: Delayed Recall	Memory. Assesses only one domain of function, but in	Learning ability, school performance

		considerable depth.	
New Zealand	Test of Language Development (TOLD).	Language development. Identifies children who are significantly below their peers in language proficiency.	Literacy skills, learning, school performance.
	Wechsler Intelligence Scale for Children-Revised Performance IQ	Performance IQ e.g., visuospatial, sustained attention, sequential memory. Assesses learning style to determine relative strengths and weaknesses, levels of cognitive functioning, assists in the determination of a learning disability.	Learning, school performance.
	Wechsler Intelligence Scale fro Children-Revised Full-Scale IQ	Full-scale IQ e.g., performance IQ plus verbal processing and expressive vocabulary.	Learning, school performance.
	McCarthy Perceptual Performance	Measures cognitive performance such as performance IQ, visuospatial, memory. Visuospatial perception is the ability to reach for objects in space and to shift our gaze from one point to another.	Learning, school performance.
	McCarthy Motor Test	Measures gross and fine motor skills.	Difficulty with fine motor skills such as writing.

Societal and Economic Impacts of Methylmercury Exposure

The exposure of the developing child to methylmercury may well translate into lifelong impacts on brain function. While no one can say for certain how many children will suffer neurodevelopmental impairments from methylmercury exposure, EPA has indicated that 1 in 6 women of childbearing age has mercury levels in her blood above EPA's current health threshold.[18] Nationally, this means that as many as 630,000 of the four million babies born each year are at risk of developmental problems due to mercury exposure in utero.[19]

What do these staggering numbers mean for childhood development, for our education system and for our society? Developmental and learning disabilities, including loss of IQ points, have negative impacts not only on individuals, but also have long-term consequences for the population and society as a whole.[20] Chemical contamination of the brain – the ultimate pollution – affects not only the educational attainment, economic performance and income of the individual, but it also has an impact on the performance of the economy as a whole through its affect of society's potential production and rate of technical progress and overall productivity.[21]

Lowered IQ has a documented relationship with economic outcomes such as lifetime earnings.[22] Even small decrements in IQ have been linked with lower wages and earnings. Two recent studies have attempted to calculate the societal cost of methylmercury exposure in the U.S and the related economic benefits of reducing such exposure. The Center for Children's Health and the Environment at the Mt. Sinai School of Medicine concluded that exposure to methylmercury causes lifelong loss of intelligence in hundreds of thousands of American babies born each year, and that this loss of intelligence exacts a significant economic cost to American society - a cost that is estimated to be in the hundreds of million dollars each year.[23]

In a different study, the Northeast States for Coordinated Air Use Management (NESCAUM) in collaboration with the Harvard School of Public Health quantified how decreasing mercury emissions from coal-fired power plants would result in less methylmercury exposure and consequently, IQ point gains for the population of children born each year.[24] According to this study, a 70% decrease in coal-fired power plant mercury emissions by 2018 would result in benefits to society of between $119 million to $288 million every year. Consequently, a reduction in emissions of more than 70% would result in even greater benefits. Extrapolating these results, we estimate that a 90% reduction in emissions would result in benefits to society worth more than $370 million per year.

Effects on IQ however, may be just the tip of the iceberg. As researchers point out, IQ effects may be the easiest to quantify and put a dollar value on, but they may not be the most serious in terms of life and career outcomes. Toxicants like methylmercury that affect the nervous system, alter a person's ability to plan, organize and initiate ideas and may induce problems with attention, distractibility, impulsive behavior and inability to handle stress and disappointments. These effects could be far more serious with respect to success in school and life.[25]

According to the Learning Disabilities Association of America, 15 percent of the U.S. population, or one in seven Americans, has some type of learning disability. Among school-age children, more than 6 percent are currently receiving special education services because of learning disabilities – that's almost 3 million students![26]

In 1999-2000, the cost of educating students with disabilities represented over 21 percent of the spending on all elementary and secondary educational services in the U.S. According to the Special Education Expenditure Project:[27]

- In 1999-2000, the total spending to provide a combination of regular and special education services to students with disabilities amounted to $77.3 billion, or an average of $12,474 per student.
- The additional expenditure to educate the average student with a disability is estimated to be about $5,918 per student each year, or 1.9 times the costs of educating a student without special needs.
- Federal funding to local education agencies (including Medicaid funds) covers only 12 percent of the additional expenditure on special education students.

It is difficult to estimate the cost of learning disabilities due to methylmercury exposure. Of the 630,000 babies born each year at risk for developmental effects due to mercury exposure, researchers don't know how many children will actually suffer adverse effects because of different exposure levels and the timing of exposure relative to brain development in individual children. However, in its review of methylmercury effects, the National Research Council concluded that more than 60,000 children in the U.S. each year - those born to mothers with the highest fish consumption during pregnancy - will likely struggle to keep up in a normal classroom or may require remedial classes or special education.[28] Assuming 60,000 students at each grade level (K-12) at cost of $5,918 per student per year, one estimate of special education costs for methylmercury exposure is $4.6 billion per year. This estimate is illustrative of the potential enormous costs of special education due to mercury exposure during pregnancy.

Stopping Mercury Pollution at the Source

To decrease human exposure to methylmercury, mercury contamination in fish must be reduced. In order to reduce mercury contamination in fish, mercury emissions that deposit from the atmosphere into our waterways must be reduced. The largest and last unregulated industrial source of mercury emissions in the U.S. is coal-fired power plants. Mercury emissions from these power plants must be reduced to the maximum extent possible, as quickly as possible. The Clean Air Act requires EPA to issue "maximum achievable control technology" (MACT) standards for coal-fired power plants. Power plants would have to comply with these rules in 2008. Independent tests indicate that 90 percent removal of mercury will be achievable and affordable by that time.[29,30] In fact, some power plants are reducing their emissions by more than 90 percent right now.[31] There is no need to wait 15 or more years to reduce mercury pollution from these sources as new industry-supported legislative proposals would allow.

Strong regulations must require that each power plant reduce its emissions and not allow the "trading" of mercury emission "credits". Emissions trading means that some power plants may not have to reduce their emissions at all. Instead, they could buy mercury emission "credits" from other power plants and do nothing to stop contamination of local lakes and streams. Some plants could even *increase* their mercury emissions. Because mercury trading could lead to toxic hotspots where mercury contamination increases, EPA must bar trading in mercury emissions. A final regulation to address mercury pollution from power plants is expected from EPA in 2005. EPA's 2004 proposed regulation generated enormous public debate, and was criticized by children's health advocates and the public health community as weak and not protective enough of the health risks to unborn babies, infants, and children.

In addition, because of the global circulation of mercury in the atmosphere, a binding global treaty that requires all nations to reduce their mercury use and emissions must be negotiated. This global agreement must complement - not replace - our national efforts to reduce mercury emissions in this country as required by the Clean Air Act. During February 2005 meetings of the United Nations Environment Programme (UNEP), the United States delegation opposed a proposal by several governments, including members of the European Union, for a legally binding pact to ban mercury. Instead, U.S. diplomats called for voluntary public-private partnerships to reduce mercury levels. The option of a binding treaty will be revisited by the UNEP governing council in 2007. The U.S. must step up in a leadership role and commit to reducing mercury use and emissions at home and abroad.

Endnotes

[1] U.S. EPA. Health Effects of Mercury and Mercury Compounds. Volume V of Mercury Study Report to Congress. 1997f., (EPA-452/R-97-007).
[2] Committee on the Toxicological Effects of Methylmercury. Board on Environmental Studies and Toxicology. Commission on Life Sciences. National Research Council. Toxicological Effects of Methylmercury, 2000. National Academy Press. Online. Available: http://www.nap.edu/books/0309071402/html/
[3] Committee on the Toxicological Effects of Methylmercury. Board on Environmental Studies and Toxicology. Commission on Life Sciences. National Research Council. Toxicological Effects of Methylmercury, 2000. National Academy Press. Online. Available: http://www.nap.edu/books/0309071402/html/
[4] U.S. EPA, 1997b. Mercury Study Report to Congress, Volume VII: Characterization of Human and Wildlife Risks from Mercury Exposure in the United States. EPA-452/R-97-009
[5]U.S. EPA, 1997b. Mercury Study Report to Congress, Volume VII: Characterization of Human and Wildlife Risks from Mercury Exposure in the United States. EPA-452/R-97-009
[6] U.S. EPA, 1997b. Mercury Study Report to Congress, Volume VII: Characterization of Human and Wildlife Risks from Mercury Exposure in the United States. EPA-452/R-97-009
[7] http://www.epa.gov/waterscience/fish/advisories/factsheet.pdf
[8] http://www.epa.gov/waterscience/fish/advisories/factsheet.pdf
[9] http://www.fda.gov
[10] Centers for Disease Control, January 2003. Second National Report on Human Exposure to Environmental Chemicals.
[11] Derived by the Clean Air Task Force from 2000 census data and fertility data from the National Center for Health Statistics.
[12] Rice, D.C., Schoeny, R., and K. Mahaffey, 2003. Methods and derivation of a reference dose for methylmercury by the U.S. EPA. Risk Analysis, Vol. 23, No. 1, pp. 107-115.
[13]Rice, D.C., Schoeny, R., and K. Mahaffey, 2003. Methods and derivation of a reference dose for methylmercury by the U.S. EPA. Risk Analysis, Vol. 23, No. 1, pp. 107-115.
[14] Committee on the Toxicological Effects of Methylmercury. Board on Environmental Studies and Toxicology. Commission on Life Sciences. National Research Council. Toxicological Effects of Methylmercury, 2000. National Academy Press. Online. Available: http://www.nap.edu/books/0309071402/html/
[15]Rice, D.C., Schoeny, R., and K. Mahaffey, 2003. Methods and derivation of a reference dose for methylmercury by the U.S. EPA. Risk Analysis, Vol. 23, No. 1, pp. 107-115.
[16] Committee on the Toxicological Effects of Methylmercury. Board on Environmental Studies and Toxicology. Commission on Life Sciences. National Research Council. Toxicological Effects of Methylmercury, 2000. National Academy Press. Online. Available: http://www.nap.edu/books/0309071402/html/
[17] Murata, K., P. Weihe, E. Buditz-Jorgensen, P.J. Jorgensen, P. Grandjean,2004. Delayed brainstem auditory evoked potential latencies in 14-yearpold children exposed to methylmercury. Journal of Pediatrics, February, pp. 177-183.
[18] Centers for Disease Control, January 2003. Second National Report on Human Exposure to Environmental Chemicals.
[19] Derived by the Clean Air Task Force from 2000 census data and fertility data from the National Center for Health Statistics.
[20] Muir, T. and M. Zegarac, 2001. Societal costs of exposure to toxic substances: economic and health costs of four case studies that are candidates for environmental causation. Envr. Health Perspect. Volume 109, Sup. 6, pp. 885-903. December.
[21]Muir, T. and M. Zegarac, 2001. Societal costs of exposure to toxic substances: economic and health costs of four case studies that are candidates for environmental causation. Envr. Health Perspect. Volume 109, Sup. 6, pp. 885-903. December.
[22]Muir, T. and M. Zegarac, 2001. Societal costs of exposure to toxic substances: economic and health costs of four case studies that are candidates for environmental causation. Envr. Health Perspect. Volume 109, Sup. 6, pp. 885-903. December.
[23] Trasande, L., P. Landrigan and C. Schechter, 2005. Public health and economic consequences of methylmercury toxicity to the developing brain. *Environ Health Perspect*: doi:10.1289/ehp.7743. [Online 28 February 2005]

http://ehp.niehs.nih.gov/docs/2005/7743/abstract.html

[24] Northeast States for Coordinated Air Use Management. Economic valuation of human health benefits of controlling mercury emissions from U.S. coal-fired power plants. February, 2005.

[25]Muir, T. and M. Zegarac, 2001. Societal costs of exposure to toxic substances: economic and health costs of four case studies that are candidates for environmental causation. Envr. Health Perspect. Volume 109, Sup. 6, pp. 885-903. December.

[26] Learning Disabilities Association of America. http://ldanatl.org

[27] Special Education Expenditure Project. Total Expenditures for Students with Disabilities: 1999-2000: Spending Variation by Disability. Report 5, June 2003. Center for Special Education Finance.

[28] Committee on the Toxicological Effects of Methylmercury. Board on Environmental Studies and Toxicology. Commission on Life Sciences. National Research Council. Toxicological Effects of Methylmercury, 2000. National Academy Press. Online. Available: http://www.nap.edu/books/0309071402/html/

[29] ADA Environmental Solutions. Progress made during 2004 on activated carbon injection technology for control of mercury from coal-fired boilers. Submitted to the U.S. EPA, December 30, 2004. Docket# OAR-2002-0056.

[30] Sorbent Technologies, Inc. The current state of activated carbon technology. Submitted to the U.S. EPA, December 30, 2004. Docket# OAR-2002-0056.

[31] U.S. EPA, 2003. Performance and cost of mercury and multipollutant emission control technology applications on electric utility boilers. October. EPA-600/R-03-110.

Great Falls, Tribune
2/9/07

One in 150 children has autism disorder

■ Reason for increase not yet understood

USA Today

Autism disorders are more common than previously known, affecting an average of one in 150 children, according to a new federal report.

Earlier estimates placed the rate at one in 166 children.

But Catherine Rice, lead author on the new report from the Centers for Disease Control and Prevention released Thursday, said the previous estimates were based on a variety of studies, most of them smaller than these latest ones, involving different study methods from several countries.

Facts about Autism Spectrum Disorder:

■ The category includes autism, PDD-NOS (pervasive developmental disorder not otherwise specified) and Asperger's disorder.

■ All are marked by problems with language and communication and often are accompanied by repetitive or unusual behaviors. ASDs begin before age 3, but might not be diagnosed until later. The causes are still being debated.

— USA Today

For the new report, the CDC drew on information gathered in 2000 and 2002 by a multistate surveillance network on 8-year-olds identified as having an Autism Spectrum Disorder (ASD).

The 2000 study involved sites in six states and 1,252 children with ASD.

Researchers found a prevalence ranging from 4.5 children in 1,000 in West Virginia up to 9.9 in 1,000 in New Jersey, with an average 6.7 in 1,000.

The 2002 study involved 2,685 kids with ASD at sites in 14 states and found autism prevalence ranged from a low of 3.3 in 1,000 children in Alabama to 10.6 in 1,000 in New Jersey, an average of 6.6 in 1,000.

Because ASDs vary and some states have better diagnosis and tracking, estimates differ, Rice said.

"Our estimates are becoming better and more consistent, though we can't yet tell if there is a true increase in ASDs or if the changes are the result of our better studies," CDC Director Julie Gerberding said in a statement.

Peter Bell, president of Cure Autism Now, said the report provides "confirmation of our worst fears. ... Every two or three years, we're given an estimate that is higher than the previous one."

Public Health and Economic Consequences of Methyl Mercury Toxicity to the Developing Brain

Leonardo Trasande,[1,2,3,4] Philip J. Landrigan,[1,2] and Clyde Schechter[5]

[1]Center for Children's Health and the Environment, Department of Community and Preventive Medicine, and [2]Department of Pediatrics, Mount Sinai School of Medicine, New York, New York, USA; [3]Division of General Pediatrics, Children's Hospital, Boston, Massachusetts, USA; [4]Department of Pediatrics, Harvard Medical School, Boston, Massachusetts, USA; [5]Department of Family Medicine, Albert Einstein College of Medicine, Bronx, New York, USA

Methyl mercury is a developmental neurotoxicant. Exposure results principally from consumption by pregnant women of seafood contaminated by mercury from anthropogenic (70%) and natural (30%) sources. Throughout the 1990s, the U.S. Environmental Protection Agency (EPA) made steady progress in reducing mercury emissions from anthropogenic sources, especially from power plants, which account for 41% of anthropogenic emissions. However, the U.S. EPA recently proposed to slow this progress, citing high costs of pollution abatement. To put into perspective the costs of controlling emissions from American power plants, we have estimated the economic costs of methyl mercury toxicity attributable to mercury from these plants. We used an environmentally attributable fraction model and limited our analysis to the neurodevelopmental impacts—specifically loss of intelligence. Using national blood mercury prevalence data from the Centers for Disease Control and Prevention, we found that between 316,588 and 637,233 children each year have cord blood mercury levels > 5.8 µg/L, a level associated with loss of IQ. The resulting loss of intelligence causes diminished economic productivity that persists over the entire lifetime of these children. This lost productivity is the major cost of methyl mercury toxicity, and it amounts to \$8.7 billion annually (range, \$2.2–43.8 billion; all costs are in 2000 US\$). Of this total, \$1.3 billion (range, \$0.1–6.5 billion) each year is attributable to mercury emissions from American power plants. This significant toll threatens the economic health and security of the United States and should be considered in the debate on mercury pollution controls. *Key words:* children's health, cognitive development, cord blood, electrical generation facilities, environmentally attributable fraction, fetal exposure, lost economic productivity, mercury, methyl mercury, power plants. *Environ Health Perspect* 113:590–596 (2005). doi:10.1289/ehp.7743 available via *http://dx.doi.org/* [Online 28 February 2005]

Mercury is a ubiquitous environmental toxicant (Goldman et al. 2001). It exists in three forms, each of which possesses different bioavailability and toxicity: the metallic element, inorganic salts, and organic compounds (methyl mercury, ethyl mercury, and phenyl mercury) (Franzblau 1994). Although volcanoes and other natural sources release some elemental mercury to the environment, anthropogenic emissions from coal-fired electric power generation facilities, chloralkali production, waste incineration, and other industrial activities now account for approximately 70% of the 5,500 metric tons of mercury that are released into the earth's atmosphere each year [United Nations Environmental Programme (UNEP) 2002]. Elemental mercury is readily aerosolized because of its low boiling point, and once airborne it can travel long distances to eventually deposit into soil and water. In the sediments of rivers, lakes, and the ocean, metallic mercury is transformed within microorganisms into methyl mercury (Guimaraes et al. 2000). This methyl mercury biomagnifies in the marine food chain to reach very high concentrations in predatory fish such as swordfish, tuna, king mackerel, and shark (Dietz et al. 2000; Gilmour and Riedel 2000; Mason et al. 1995; Neumann and Ward 1999). Consumption of contaminated fish is the major route of human exposure to methyl mercury.

The toxicity of methyl mercury to the developing brain was first recognized in the 1950s in Minamata, Japan, where consumption of fish with high concentrations of methyl mercury by pregnant women resulted in at least 30 cases of cerebral palsy in children; exposed women were affected minimally if at all (Harada 1968). A similar episode followed in 1972 in Iraq when the use of a methyl mercury fungicide led to poisoning in thousands of people (Bakir et al. 1973); again, infants and children were most profoundly affected (Amin-Zaki et al. 1974, 1979). The vulnerability of the developing brain to methyl mercury reflects the ability of lipophilic methyl mercury to cross the placenta and concentrate in the central nervous system (Campbell et al. 1992). Moreover, the blood–brain barrier is not fully developed until after the first year of life, and methyl mercury can cross this incomplete barrier (Rodier 1995).

Three recent, large-scale prospective epidemiologic studies have examined children who experienced methyl mercury exposures *in utero* at concentrations relevant to current U.S. exposure levels. The first of these studies, a cohort in New Zealand, found a 3-point decrement in the Wechsler Intelligence Scale-Revised (WISC-R) full-scale IQ among children born to women with maternal hair mercury concentrations > 6 µg/g (Kjellstrom et al. 1986, 1989). A second study in the Seychelles Islands in the Indian Ocean found only one adverse association with maternal hair mercury concentration among 48 neurodevelopmental end points examined (prolonged time to complete a grooved pegboard test with the nonpreferred hand) (Myers et al. 2003). However, the grooved pegboard test was one of the few neurobehavioral instruments in the Seychelles study not subject to the vagaries of translation that can degrade the validity of culture-bound tests of higher cognitive function when they are applied in developing nations (Landrigan and Goldman 2003). A third prospective study in the Faroe Islands, a component of Denmark inhabited by a Scandinavian population in the North Atlantic, has followed a cohort of children for 14 years and collected data on 17 neurodevelopmental end points, as well as on the impact of methyl mercury on cardiovascular function. The Faroes researchers found significant dose-related, adverse associations between prenatal mercury exposure and performance on a wide range of memory, attention, language, and visual-spatial perception tests (Grandjean et al. 1997). The significance of these associations remained evident when

Address correspondence to L. Trasande, Center for Children's Health and the Environment, Department of Community and Preventive Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Place, Box 1057, New York, NY 10029 USA. Telephone: (212) 241-8029. Fax: (212) 996-0407. E-mail: leo.trasande@mssm.edu

We are grateful for the helpful advice of P. Grandjean, E. Budtz-Jorgensen, D. Laraque, P. Leigh, and J. Palfrey as well as to D.P. Rice and W. Max for their assistance with lifetime earnings estimates.

This research was supported by a grant from the National Institute of Environmental Health Sciences through the Superfund Basic Research Program (PT42ES07384) to Mount Sinai School of Medicine, and by the Jenifer Altman Foundation, Physicians for Social Responsibility, and the Rena Shulsky Foundation.

The authors declare they have no competing financial interests.

Received 10 November 2004; accepted 28 February 2005.

blood levels of polychlorinated biphenyls, which are known developmental neurotoxicants (Jacobson and Jacobson 1996), were included in the analysis (Budtz-Jorgensen et al. 2002; Steuerwald et al. 2000). Methyl mercury exposure was also associated with decreased sympathetic- and parasympathetic-mediated modulation of heart rate variability (Grandjean et al. 2004) and with persistent delays in peaks I–III brainstem evoked potentials (Murata et al. 2004).

good

An assessment of these three prospective studies by the National Academy of Sciences (NAS) (National Research Council 2000) concluded that there is strong evidence for the fetal neurotoxicity of methyl mercury, even at low concentrations of exposure. Moreover, the NAS opined that the most credible of the three prospective epidemiologic studies was the Faroe Islands investigation. In recommending a procedure for setting a reference dose for a methyl mercury standard, the NAS chose to use a linear model to represent the relationship between mercury exposure and neurodevelopmental outcomes, and based this model on the Faroe Islands data. The NAS found that the cord blood methyl mercury concentration was the most sensitive biomarker of exposure *in utero* and correlated best with neurobehavioral outcomes. The NAS was not deterred by the apparently negative findings of the Seychelles Islands study, which it noted was based on a smaller cohort than the Faroe Islands investigation and had only 50% statistical power to detect the effects observed in the Faroes (National Research Council 2000).

Since January 2003, the issue of early life exposure to methyl mercury has become the topic of intense debate after the U.S. Environmental Protection Agency (EPA) announced a proposal to reverse strict controls on emissions of mercury from coal-fired power plants. This proposed "Clear Skies Act" would slow recent progress in controlling mercury emission rates from electric generation facilities and would allow these releases to remain as high as 26 tons/year through 2010 (U.S. EPA 2004a). By contrast, existing protections under the Clean Air Act will limit mercury emissions from coal-fired power plants to 5 tons/year by 2008 (U.S. EPA 2004b). The U.S. EPA's technical analyses in support of "Clear Skies" failed to incorporate or quantify consideration of the health impacts resulting from increased mercury emissions (U.S. EPA 2004c). After legislative momentum for this proposal faded, the U.S. EPA proposed an almost identical Utility Mercury Reductions Rule, which again failed to examine impacts on health. The U.S. EPA issued a final rule on 15 March 2005 (U.S. EPA 2005).

To assess the costs that may result from exposure of the developing brain to methyl mercury, we estimated the economic impact of anthropogenic methyl mercury exposure in the 2000 U.S. birth cohort. We calculated the fraction of this cost that could be attributed to mercury emitted by American electric power generation facilities.

Materials and Methods

Environmentally attributable fraction model. To assess the disease burden and the costs due to methyl mercury exposure, we used an environmentally attributable fraction (EAF) model. The EAF approach was developed by the Institute of Medicine (IOM) to assess the "fractional contribution" of the environment to causation of illness in the United States (IOM 1981), and it has been used to assess the costs of environmental and occupational disease (Fahs et al. 1989; Leigh et al. 1997). It was used recently to estimate the environmentally attributable costs of lead poisoning, asthma, pediatric cancer, and neurodevelopmental disabilities in American children (Landrigan et al. 2002). The EAF is defined by Smith et al. (1999) as "the percentage of a particular disease category that would be eliminated if environmental risk factors were reduced to their lowest feasible concentrations." The EAF is a composite value and is the product of the prevalence of a risk factor multiplied by the relative risk of disease associated with that risk factor. Its calculation is useful in developing strategies for resource allocation and prioritization in public health. The general model developed by the IOM and used in the present analysis is the following:

$$\text{Costs} = \text{disease rate} \times \text{EAF} \times \text{population size} \times \text{cost per case}$$

"Cost per case" refers to discounted lifetime expenditures attributable to a particular disease, including direct costs of health care, costs of rehabilitation, and lost productivity. "Disease rate" and "population size" refer, respectively, to the incidence or prevalence of a disease and the size of the population at risk.

In applying the EAF model, we first reviewed the adverse effects of methyl mercury exposure. We then estimated the costs of those effects and subsequently applied a further fraction to parse out the cost of anthropogenic methyl mercury exposure resulting from emissions of American electrical generation facilities.

Toxic effects of methyl mercury exposure. The NAS found neurodevelopmental effects in the children of women who had consumed fish and seafood during pregnancy to be the most important and best-studied end point for methyl mercury toxicity. Although the NAS identified other potentially significant toxicities resulting from methyl mercury exposure, such as nephrotoxicity and carcinogenicity, those effects were less well characterized (National Research Council 2000). We therefore limited our analysis to the neurodevelopmental impact of methyl mercury toxicity.

There is no evidence to date validating the existence of a threshold blood mercury concentration below which adverse effects on cognition are not seen. The U.S. EPA has, however, set a benchmark dose level (BMDL) for cord blood mercury dose concentration of 58 μg/L. This level that corresponds to the lower limit of the 95% confidence interval for the concentration at which there is a doubling in the Faroes study in the prevalence of test scores (5–10%) in the clinically subnormal range for the Boston Naming Test (Rice et al. 2003). It is important to note that this is not a concentration below which no observed adverse effects were found. The Faroes and New Zealand cohorts both support the conclusion that developmental effects become apparent at levels of approximately 1 ppm mercury in hair, or 5.8 μg/L in cord blood (Grandjean et al. 1997; Kjellstrom et al. 1986, 1989). The Faroes study also found that effects on delayed brainstem auditory responses occurred at much lower exposure concentrations (Murata et al. 2004). In its report, the NAS concluded that the likelihood of subnormal scores on neurodevelopmental tests after *in utero* exposure to methyl mercury increased as cord blood concentrations increased from levels as low as 5 μg/L to the BMDL of 58 μg/L (National Research Council 2000). In light of those findings, we decided in this analysis to apply a no adverse effect level of 5.8 μg/L, the lowest level at which adverse neurodevelopmental effects were demonstrated in the cohort studies.

Recent data suggest that the cord blood mercury concentration may on average be 70% higher than the maternal blood mercury concentration (Stern and Smith 2003), and a recent analysis suggests that a modification of the U.S. EPA reference dose for methyl mercury be made to reflect a cord blood:maternal blood ratio that is > 1 (Stern 2005). If the developmental effects of mercury exposure do, in fact, begin at 5.8 μg/L in cord blood, as suggested by the Faroes (Grandjean et al. 1997) and New Zealand (Kjellstrom et al. 1986, 1989) data and by the NAS report (National Research Council 2000), then effects would occur in children born to women of childbearing age with blood mercury concentrations ≥ 3.41 (ratio, 5.8:1.7) μg/L. National population data from the 1999–2000 National Health and Nutrition Examination Survey (NHANES) found that 15.7% of American women of childbearing age have total blood mercury concentrations ≥ 3.5 μg/L (Mahaffey et al. 2004).

To compute IQ decrements in infants that have resulted from these elevated maternal mercury exposures, we used published data on percentages of women of childbearing age with mercury concentrations ≥ 3.5, 4.84, 5.8, 7.13, and 15.0 µg/L. We assumed conservatively that all mercury concentrations within each of the segments of the distribution were at the lower bound of the range. We assumed that the probability of giving birth to a child did not correlate with mercury level in a woman of childbearing age. In our base case analysis, we calculated economic costs assuming that children born to women with mercury concentrations 3.5–4.84 µg/L suffer no loss in cognition, and that successive portions of the birth cohort experience loss of cognition associated with cord blood levels of 8.2, 9.9, 12.1, and 25.5 µg/L, respectively.

Recently, the Faroes researchers reviewed their cohort data and found fetal blood mercury concentrations to be only 30% higher than maternal blood concentrations (Budtz-Jorgensen et al. 2004). In light of these findings and to avoid overestimation of the magnitude of impacts, we chose not to include children born to mothers with blood mercury concentrations between 3.5 and 4.84 µg/L in our base case analysis.

To assess the impact on our findings of a range of various possible ratios between maternal and cord blood mercury concentrations, we conducted a sensitivity analysis. In this analysis, we set as a lower bound for our estimate the costs to children with estimated cord blood concentrations ≥ 5.8 µg/L (assuming a cord:maternal blood ratio of 1) and assumed no IQ impact < 4.84 µg/L (assuming a cord:maternal blood ratio of 1.19). This estimate assumed no loss of cognition to children born to women with mercury concentration < 5.8 µg/L and assumed that subsequent portions of the birth cohort experienced cord blood mercury concentrations of 5.8, 7.13, and 15 µg/L, respectively. To estimate economic costs in this scenario, we calculated no costs for children with blood mercury concentrations < 4.84 µg/L. We calculated costs resulting from an incremental increase in blood mercury concentration from 4.84 to 5.8 µg/L in the percentage of the population with blood mercury levels between 5.8 and 7.13 µg/L, and added those costs to the costs resulting from increases from 4.84 to 7.13 µg/L and 4.84 to 15 µg/L in the percentages of the population with concentrations between 7.13 and 15 µg/L and > 15 µg/L, respectively. The result of this calculation is expressed in our analysis as a lower bound for the true economic cost of methyl mercury toxicity to the developing brain.

Impact of methyl mercury exposure on IQ. The Faroes study found that a doubling of mercury concentration was associated with adverse impacts on neurodevelopmental tests ranging from 5.69–15.93% of a standard deviation (Grandjean et al. 1999). Assuming that IQ is normally distributed with a standard deviation of 15 points, a doubling of mercury concentration would be associated with a decrement ranging from 0.85 to 2.4 IQ points. The Faroes researchers used a structural equation analysis to produce estimates of impact of methyl mercury on verbal and motor function at 7 years of age and found an association between a doubling of blood mercury and loss of 9.74% of a standard deviation on motor function and of 10.45% of a standard deviation on verbal function (Budtz-Jorgensen et al. 2002). This analysis suggests that a doubling in mercury concentration produces a decrement of approximately 10% of a standard deviation, or 1.5 IQ points. In the New Zealand study (Kjellstrom et al. 1986, 1989), the average WISC-R full-scale IQ for the study population ($n = 237$) was 93. In the group with maternal hair mercury > 6 µg/g (~ 4-fold higher than in the study population, $n = 61$), the average was 90 (Kjellstrom et al. 1989). This finding further supports our use of a loss of 1.5 IQ points for each doubling in our base case analysis. Confounders such as polychlorinated biphenyls did not cause significant confounding of the data in the Faroe Islands study (Budtz-Jorgensen et al. 2002; Steuerwald et al. 2000). As a conservative measure, we nonetheless chose to set as outer bounds for the impact on intelligence of methyl mercury exposure a range of IQ decrements from 0.85 to 2.4 IQ points per doubling, as described by the Faroes researchers (Jorgensen et al. 2004). In applying the EAF methodology, we assume that the relationship between cord blood mercury and IQ is relatively linear over the range of exposures studied (> 5.8 µg/L).

In our sensitivity analysis, we used the same linear dose–response model that was selected by the National Research Council in setting a reference dose for mercury exposure (National Research Council 2000). The Faroes researchers found that, for those children whose mothers had hair mercury concentrations < 10 µg/g, a 1-µg/L increase of cord blood mercury concentration was associated with adverse impacts on neurodevelopmental tests ranging from 3.95 to 8.33% of a standard deviation, or 0.59–1.24 IQ points (average = 0.93 IQ points) (Jorgensen et al. 2004). We also varied the cord:maternal blood mercury ratio from 1 to 1.7 in calculating IQ impact from the linear model as part of our sensitivity analysis. As an upper bound to our cost estimate using the logarithmic model, we calculated the economic cost assuming that children born to women with mercury concentrations 3.5–4.84 µg/L suffer no loss in cognition and that successive portions of the birth cohort experience losses of cognition of 1.21, 1.84, 2.55, and 5.13 IQ points, respectively. The lower-bound estimate assumed that children born to women with mercury concentrations 4.84–5.8 µg/L suffer no loss in cognition and that successive portions of the birth cohort experience losses of cognition of 0.22, 0.48, and 1.39 IQ points.

As an upper bound to our cost estimate using the linear model, we calculated the economic cost assuming that children born to women with mercury concentrations 3.5–4.84 µg/L suffer no loss in cognition and that successive portions of the birth cohort experience losses of cognition of 3.01, 5.04, 7.84, and 24.43 IQ points, respectively. The lower-bound estimate assumed that children born to women with mercury concentrations 4.84–5.8 µg/L suffer no loss in cognition and that successive portions of the birth cohort experience losses of cognition of 0.56, 1.35, and 5.99 IQ points.

Calculation of economic costs of IQ loss. To estimate the costs associated with the cognitive and behavioral consequences of mercury exposure, we relied on an economic forecasting model developed by Schwartz et al. (1985), and we applied this model to NHANES data on prevalence of mercury exposure in women of childbearing age (Schober et al. 2003; Schwartz et al. 1985). In this model, lead concentrations are assumed on the basis of work by Salkever (1995) to produce a dose-related decrement in IQ score. Those decrements in IQ are, in turn, associated with lower wages and diminished lifetime earning power. Salkever used three regression techniques to derive direct and indirect relationships among IQ, schooling, probability of workforce participation, and earnings. He estimated a percentage in lost earnings per IQ point from the percent loss of earnings for each microgram per deciliter increase in blood lead level. Salkever found a 0.473 point decrement in lost lifetime earnings for each microgram per deciliter increase among men and a 0.806 point decrement for each microgram per deciliter increase among women (Salkever 1995). Using Schwartz's (1994) estimate that 0.245 IQ points are lost for each microgram per deciliter increase in blood lead, Salkever (1995) estimated a percentage loss in lifetime earnings per IQ point among men (1.931%) and women (3.225%). We assume that this relationship remains linear across the population range of IQ.

Assuming an annual growth in productivity of 1% and applying a 3% real discount rate, the present value of lifetime expected earnings is $1,032,002 for a boy born in 2000 and $763,468 for a girl born in the same year (Max et al. 2002). The costs of the diminution in this earning power were calculated for the 2000 American birth cohort, using available data on the number of male and female births in 2000 [Centers for Disease Control and Prevention (CDC) 2002a]. We diminished our cost estimate by

0.69%, the infant mortality rate in 2000, to account for those children for whom methyl mercury exposure is unlikely to result in diminished economic productivity (CDC 2002b).

American sources of mercury emission. Mercury emissions result from anthropogenic as well as from natural sources, and we limited our analysis to methyl mercury derived from anthropogenic sources. The UNEP recently estimated that anthropogenic uses account for 70% of the 5,500 tons of mercury released into the earth's atmosphere worldwide (UNEP 2002). Therefore, to limit our analysis to anthropogenic mercury, we applied a 70% factor to convert the cost of lost economic productivity resulting from methyl mercury exposure to the cost attributable to anthropogenic methyl mercury exposure.

We next parsed out the proportion of anthropogenic methyl mercury in fish that arises from American sources and then isolated the subset of that proportion that is emitted by coal-fired electrical generating plants. In 1995, the most recent year for which federal data on the relative deposition of mercury from American and other global sources are available, 158 tons of mercury were emitted to the atmosphere by American anthropogenic sources. Fifty-two (33%) of those 158 tons were deposited in the lower 48 states, whereas the remaining two-thirds were added to the global reservoir (U.S. EPA 2004d). Also in 1995, an additional 35 tons of mercury from the global reservoir were deposited in the United States. Therefore, a total of 87 total tons of mercury were deposited in the United States in that year, of which 60% (52 of 87) were attributable to American anthropogenic sources (U.S. EPA 1996, 1997). This mercury would have been available to bioaccumulate in the marine and aquatic food chains and to enter American freshwater and saltwater fish.

Further complicating our calculations is the fact that not all of the fish sold in America is from American sources. Of the 10.4 billion pounds of edible fish supplied in the United States in 2002, 4.4 billion (42%) are imported from sources outside of the United States (National Marine Fisheries Service 2002). Because U.S. emissions account for 3% of global emissions (UNEP 2002; U.S. EPA 1996), we calculate that the mercury content of imported fish is 2% of American anthropogenic origin: 158 tons of American emissions – 52 tons of American mercury deposited on American soil = 106 tons of American mercury available to contaminate imported fish; 5,500 tons emitted globally – 87 tons deposited on American soil = 5,413 tons of mercury from all sources to contaminate imported fish; 106 tons of mercury available/5,413 tons of mercury from all sources = 0.02, or 2% of mercury in imported fish of American origin. In the remaining 58% of fish consumed in the United States, we assume that 60% of the mercury content comes from American anthropogenic sources (U.S. EPA 1996, 1997). We therefore applied a 36% factor (the weighted average of American sources of mercury content in fish, or $0.6 \times 0.58 + 0.02 \times 0.42$) to specify the economic costs of anthropogenic methyl mercury exposure attributable to American sources.

Modeling supported by the Electric Power Resource Institute (EPRI) estimates that 70% of the mercury deposited in the United States comes from foreign sources (Seigneur et al. 2004). This EPRI analysis also finds that U.S. sources are responsible for > 60% of mercury deposition in the Boston–Washington, D.C. corridor. In one of the model's selected receptor areas—Pines Lake, New Jersey—80% of the deposition originated from U.S. sources, showing that regional deposition can be higher than the 60% number we use in this analysis (Seigneur et al. 2004). In our sensitivity analysis, we varied the factor used to convert the economic cost of anthropogenic methyl mercury exposure to the economic cost attributable to American sources from 18% ($0.3 \times 0.58 + 0.02 \times 0.42$, using EPRI's modeling) to 36% (using federal data on mercury deposition) (Seigneur et al. 2004).

In 1999, the most recent year for which data on American mercury emissions are available, 48 (41%) of the 117 tons of mercury emissions from anthropogenic sources in the United States were emitted by electric power generation facilities (U.S. EPA 2003a). To calculate the economic cost of methyl mercury exposure attributable to these facilities, we applied an additional fraction of 41% in our analysis.

Results

Base-case analysis. Each year in the United States, between 316,588 (7.8% of the annual birth cohort) and 637,233 babies are born with cord blood mercury levels > 5.8 µg/L. The lower-bound estimate of 316,588 babies is based on the very conservative assumption that maternal and cord blood mercury concentrations are equal. But if the cord blood mercury concentration is on average 70% higher than the maternal blood mercury concentration, as suggested by recent research (Stern and Smith 2003), 637,233 babies, or 15.7% of the birth cohort, experience cord blood mercury levels > 5.8 µg/L. Fetal blood mercury levels > 5.8 µg/L are associated with small but significant loss of IQ. This decrement in IQ appears to be permanent and irreversible, and it adversely affects a significant portion of the annual birth cohort's economic productivity over a lifetime.

Using our base-case assumptions (impact for women with total mercury > 4.84 µg/L, cord:maternal mercury ratio = 1.7, IQ impact = 1.5 points per doubling), we calculated costs for the 405,881 children who suffer IQ decrements resulting from fetal methyl mercury exposure. Under these assumptions, 89,293 children suffered a 0.76 decrement in IQ and another 113,647 experienced a 1.15 IQ point decrement. The 5% most highly exposed children in the 2000 birth cohort suffered subclinical losses in IQ in our model ranging from 1.60 to 3.21 points. Although this diminution in intelligence is small in comparison with the loss of cognition that can result from other genetic and environmental processes, the loss resulting from methyl mercury exposure produces a significant reduction in economic productivity over a lifetime. We estimate the aggregate cost of the loss in IQ that results from exposure of American children to methyl mercury of anthropogenic origin to be $8.7 billion (all costs in 2000 US$) annually (Table 1).

Sensitivity analysis. We estimate that the cost of anthropogenic methyl mercury exposure ranges from $2.2 billion (impact only for the 316,588 children born to women with total mercury > 5.8 µg/L, IQ impact = 0.85 points per doubling) to $13.9 billion (impact for the 405,881 women with total mercury > 4.84 µg/L, IQ impact = 2.4 points per doubling). Using the linear dose–response model that was selected by the National Research Council in recommending a reference dose for mercury exposure (a model that predicts an average loss of 0.93 IQ points per 1-µg/L increase in mercury concentration) (Jorgensen et al. 2004; National Research Council 2000), we find that the environmentally attributable cost of methyl mercury exposure is $32.9 billion, assuming a cord:maternal blood mercury ratio of 1.7. Employing a linear model and assuming that the true loss in IQ resulting from a 1-µg/L increase in blood mercury ranges from 0.59 to 1.24 points, we find that the outer bounds of our estimate range from $7.0 billion (impact only for women with total mercury > 5.8 µg/L, IQ impact = 0.59 points per µg/L increase, cord:maternal mercury ratio = 1) to $43.8 billion (impact for women with total mercury > 4.84 µg/L, IQ impact = 1.24 points for each microgram per deciliter increase, cord:maternal mercury ratio = 1.7) (Table 2).

Sources of costs. After applying the 36% fraction to restrict our analysis to American anthropogenic sources, we estimate that the attributable cost of methyl mercury exposure to the developing fetus from American anthropogenic sources is $3.1 billion annually, using the logarithmic model developed by the Faroes researchers (Grandjean et al. 1999; Jorgensen et al. 2004) and assuming a 1.5-point IQ impact for each doubling of methyl mercury exposure (Budtz-Jorgensen et al. 2002). Our sensitivity analysis, in which we also varied the attributable fraction for American sources from 18% (industry data sources) to 36% (federal data sources) (Seigneur et al. 2004; U.S. EPA 1996, 1997), suggests that the true cost of methyl mercury exposure from American emissions ranges from $0.4 to $15.8 billion annually.

To focus specifically on the costs of fetal exposure to mercury released by American coal-fired power plants, we examined the impact of the 41% of U.S. anthropogenic emissions of mercury attributable to these facilities. We estimate that the attributable cost of methyl mercury exposure from American electric generation facilities to the developing fetus is $1.3 billion. Applying our sensitivity analysis in this model, we find that the true cost of methyl mercury exposure from electric generation facilities to the American birth cohort ranges from $0.1 to $6.5 billion/year (Figure 1). Again, the major source of these costs is loss of earnings over a lifetime.

Discussion

The major findings in this analysis are *a*) that exposure to methyl mercury emitted to the atmosphere by American electric generation facilities causes lifelong loss of intelligence in hundreds of thousands of American babies born each year and *b*) that this loss of intelligence exacts a significant economic cost to American society, a cost that amounts to at least hundreds of millions of dollars each year. Moreover, these costs will recur each year with each new birth cohort as long as mercury emissions are not controlled. By contrast, the cost of installing stack filters to control atmospheric mercury emissions is a one-time expense. The high costs of *in utero* exposure to methyl mercury are due principally to the lifelong consequences of irreversible injury to the developing brain. Similar lifelong neurobehavioral consequences have been observed after exposure of the developing brain to other environmental toxicants, including lead (Baghurst et al. 1987; Bellinger 2004; Dietrich et al. 1987; Opler et al. 2004; Wasserman et al. 2000), polychlorinated biphenyls (Jacobson and Jacobson 1996), and ethanol (Lupton et al. 2004).

Because the literature has presented a range of possible consequences for methyl mercury toxicity, we have provided a range of possible public health and economic consequences. This range is meant to inform the choices that environmental and public health officials make in protecting vulnerable populations from methyl mercury exposure. Our range for the true economic costs of methyl mercury toxicity to the developing brain omits the cost of exposures to the 231,352 children born to women in 2000 with blood mercury concentrations between 3.5 and 4.84 µg/L. If the true cord blood ratio is 1.7 times the maternal blood concentration, as described in the most recent and extensive meta-analysis on the matter (Stern and Smith 2003), these children are also born with cord blood mercury concentrations above the 5.8 µg/L concentration at which adverse neurodevelopmental impact has been found. We chose not to include them in our analysis because other studies have found lower ratios and because we restricted ourselves in this analysis to the use of available, published prevalence data of maternal blood mercury concentrations. In our sensitivity analysis, we also selected low cord:maternal blood ratios so as to describe most accurately the range of values for the true cost of methyl mercury exposure to the developing fetus.

Our analysis also omits the cost of the cardiovascular impacts of mercury exposure (Grandjean et al. 2004) or the costs of mercury exposure to children in the first 2 years of postnatal life, when myelination is still continuing and the blood–brain barrier remains vulnerable to penetration by methyl mercury (Rodier 1995). We chose not to include these aspects of methyl mercury toxicity in our range of estimates at this time because there do not exist sufficient quantitative data to permit construction of a reliable model.

A limitation on our analysis is that it did not consider other societal costs beyond decreased lifetime earnings that may result from exposure of the developing brain to methyl mercury. For example, if the value of a child's social productivity is approximately $4–9 million, as suggested by studies of willingness-to-pay (WTP) estimates of a life (Viscusi and Aldy 2004), then by the WTP methodology the true cost of methyl mercury toxicity may be much higher than our estimate. We also chose not to include other noncognitive impacts. Lead, for example, has been associated with criminality and antisocial behavior (Dietrich et al. 2001; Needleman et al. 1996, 2002; Nevin 2000; Stretesky and Lynch 2001). However, because these behaviors have not been described as yet for methyl mercury, we chose not to include such costs in our estimate.

Some will argue that our range of costs fails to incorporate the role of confounding factors in quantifying the economic consequences of methyl mercury exposure. It is true that efforts

Table 1. Cost of anthropogenic mercury (Hg) exposure using a logarithmic model.

Variable	Segment of population (percentile)			
	90–92.1 Hg	92.2–94.9 Hg	95–99.3 Hg	≥ 99.4 Hg
Range of maternal total Hg concentration	4.84–5.8 µg/L	5.8–7.13 µg/L	7.13–15.0 µg/L	> 15.0 µg/L
Assumed maternal total Hg concentration	4.84	5.8	7.13	15
No effect concentration (maternal total Hg)	3.41	3.41	3.41	3.41
IQ points lost at assumed concentration	0.76	1.15	1.60	3.21
Loss of 1 IQ points = decrease in lifetime earnings				
For boys, lifetime earnings (1.931% decrease)	$1,032,002			
For girls, lifetime earnings (3.225% decrease)	$763,468			
No. of boys in birth cohort affected	45,693	58,155	91,387	12,462
No. of girls in birth cohort affected	43,601	55,492	87,201	11,891
Lost income	$1.1 billion	$2.0 billion	$4.4 billion	$1.2 billion
Total cost = $8.7 billion in each year's birth cohort				

Assumptions: EAF = 70%, main consequence = loss of IQ over lifetime.

Table 2. Sensitivity analysis: cost of anthropogenic methyl mercury exposure.

Variable	Base-case cost estimate (range)[a]
Children born to women with Hg > 4.84 µg/L, effect > 3.5 µg/L	
Logarithmic model	$8.7 billion ($4.9–13.9 billion)
Linear model, cord:maternal Hg ratio = 1.7	$32.9 billion ($20.9–43.8 billion)
Linear model, cord:maternal Hg ratio = 1	$19.3 billion ($12.3–25.8 billion)
Children born to women with > 5.8 µg/L, effect > 4.84 µg/L	
Logarithmic model	$3.9 billion ($2.2–6.3 billion)
Linear model, cord:maternal Hg ratio = 1.7	$18.7 billion ($11.9–24.9 billion)
Linear model, cord:maternal Hg ratio = 1	$11.0 billion ($7.0–14.6 billion)
Range of estimates	
Logarithmic model	$2.2–13.9 billion
Linear model	$7.0–43.8 billion

Assumptions: EAF = 70%, main consequence = loss of IQ over lifetime.
[a]Based on range of possible IQ decrement:increase cord blood mercury.

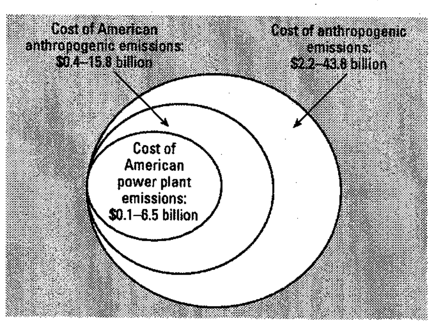


Figure 1. Portions of cost of methyl mercury exposure attributed to sources. Assumptions: 18–36% attributable to American sources; 41% of American emissions attributable to American power plants.

to delineate the potential synergistic role of methyl mercury and other chemicals in mediating neurocognitive and other effects are bedeviled by lack of knowledge about possible interactions and synergies among chemicals or between chemicals and other environmental hazards, even though the environment of a child includes mixtures of chemical and biologic toxicants. Only a study of the magnitude of the National Children's Study will facilitate simultaneous examination of the effects of multiple chemical exposures, of interactions among them, and of interactions among biologic, chemical, behavioral, and social factors (Trasande and Landrigan 2004). However, we note that loss of cognition resulting from methyl mercury exposure in the Faroe Islands study remained evident when blood levels of polychlorinated biphenyls, which are known fetal neurotoxicants (Jacobson and Jacobson 1996), were included in the analysis (Budtz-Jorgensen et al. 2002; Steuerwald et al. 2000).

We note the U.S. EPA's recent success in minimizing mercury emissions from medical waste (U.S. EPA 2004e) and municipal incinerators (U.S. EPA 2004f, 2004g), actions that resulted in a decrease in total mercury emissions by at least 80 tons per year from 1990 to 1999 (U.S. EPA 2003b). Although data are not available on blood mercury concentrations over the past decade that followed from those actions, the impact of these reductions is likely to have been substantial.

Some commentators have used data from the Seychelles study to argue that methyl mercury is not toxic to the fetus at low concentrations and to suggest that fear of mercury exposure is needlessly preventing women from ingesting fish and thus denying them access to beneficial long-chain polyunsaturated fatty acids (LCPUFAs), especially docosahexaenoic acid (DHA). We do not dispute that DHA and other LCPUFAs are important for optimal development of the fetal visual and nervous systems (Innis 1991). The human fetus has a limited ability to synthesize DHA's precursor, α-linolenic acid, and therefore it must be largely supplied from maternal sources (Carnielli et al. 1996; Larque et al. 2002; Szitanyi et al. 1999). We also note a report that associated an average monthly decline in fish consumption of 1.4 servings among Massachusetts women with a U.S. Food and Drug Administration advisory on the health risks of mercury (Oken et al. 2003). Nonetheless, the American Heart Association, a strong advocate for the cardioprotective effects of LCPUFAs, recommends that children and pregnant and lactating women avoid potentially contaminated fish (Kris-Etherton et al. 2002). Fish advisories should not recommend that consumers abstain from fish, but they should assist in choosing the best kinds of fish to eat. Lists of fish that are safe and unsafe from the perspective of mercury exposure have been published and made widely available to consumers (U.S. EPA 2004h).

Early reports of disease and dysfunction of environmental origin in children have on repeated occasions failed to produce proactive response to protect children. The long history of lead use in the United States provides a chilling reminder of the consequences of failure to act on early evidence of harm. It is important that we not repeat this sequence with mercury. Within the last century, as a result of increased industrial activity, mercury emissions worldwide have increased 2- to 5-fold, and anthropogenic emissions now surpass emissions from natural sources (Nriagu 1989).

The data from this analysis reinforce the results of recent epidemiologic studies and indicate an urgent need on economic grounds for regulatory intervention at the federal level to minimize mercury emissions. Our analysis captures the cost of methyl mercury exposure for only 1 year's birth cohort, but the cost of mercury exposure will continue to accrue in each succeeding year if power plants fail to install flue gas filters (U.S. Department of Energy 2004) or to implement other technologies to reduce mercury emissions. The cost savings from reducing mercury exposure now will provide savings in improved productivity and enhanced national security for generations to come.

References

Amin-Zaki L, Elhassani S, Majeed MA, Clarkson TW, Doherty RA, Greenwood M. 1974. Intrauterine methylmercury poisoning in Iraq. Pediatrics 54:587–595.

Amin-Zaki L, Elhassani S, Majeed MA, Clarkson TW, Greenwood M, Doherty RA. 1979. Prenatal methylmercury poisoning. Clinical observations over five years. Am J Dis Child 133:172–177.

Baghurst PA, Robertson EF, McMichael AJ, Vimpani GV, Wigg NR, Roberts RR. 1987. The Port Pirie Cohort Study: lead effects on pregnancy outcome and early childhood development. Neurotoxicology 8(3):395–401.

Bakir F, Damluji SF, Amin-Zaki L, Murtadha M, Khalidi A, al-Rawi NY, et al. 1973. Methylmercury poisoning in Iraq. Science 181:230–241.

Bellinger DC. 2004. Lead. Pediatrics 113(4 suppl):1016–1022.

Budtz-Jorgensen E, Grandjean P, Jørgensen PJ, Weihe P, Keiding N. 2004. Association between mercury concentrations in blood and hair in methylmercury-exposed subjects at different ages. Environ Res 95(3):385–393.

Budtz-Jorgensen E, Keiding N, Grandjean P, Weihe P. 2002. Estimation of health effects of prenatal methylmercury exposure using structural equation models. Environ Health 1(1):2.

Campbell D, Gonzales M, Sullivan JB Jr. 1992. Mercury. In: Hazardous Materials Toxicology—Clinical Principles of Environmental Health (Sullivan JB Jr, Krieger GR, eds). Baltimore, MD:Williams and Wilkins, 824–833.

Carnielli VP, Wattimena DJ, Luijendijk IH, Boerlage A, Degenhart HJ, Sauer PJ. 1996. The very low birth weight premature infant is capable of synthesizing arachidonic and docosahexaenoic acids from linoleic and linolenic acids. Pediatric Res 40:169–174.

CDC (Centers for Disease Control and Prevention). 2002a. National Vital Statistics System. Births: final data for 2000. Natl Vit Stat Rep 50(5):1–102.

CDC (Centers for Disease Control and Prevention). 2002b. National Vital Statistics System. Infant mortality statistics from the 2000 period linked birth/infant death data set. Natl Vit Stat Rep 50(12):1–27. Available: http://www.cdc.gov/nchs/data/nvsr/nvsr50/nvsr50_12.pdf [accessed 1 November 2004].

Dietrich KN, Krafft KM, Bornschein RL, Hammond PB, Berger O, Succop PA, et al. 1987. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. Pediatrics 80(5):721–730.

Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL. 2001. Early exposure to lead and juvenile delinquency. Neurotoxicol Teratol 23:511–518.

Dietz R, Riget F, Cleemann M, Aarkrog A, Johansen P, Hansen JC. 2000. Comparison of contaminants from different trophic concentrations and ecosystems. Sci Total Environ 245(1–3):221–223.

Fahs MC, Markowitz SB, Fischer E, Shapiro J, Landrigan PJ. 1989. Health costs of occupational disease in New York State. Am J Ind Med 16:437–449.

Franzblau A. 1994. Mercury. In: Textbook of Occupational and Environmental Medicine (Rosenstock L, Cullen M, eds). Philadelphia:W.B. Saunders, 756–759.

Gilmour CC, Riedel GS. 2000. A survey of size-specific mercury concentrations in game fish from Maryland fresh and estuarine waters. Arch Environ Contam Toxicol 39(1):53–59.

Goldman LR, Shannon MW, for the American Academy of Pediatrics Committee on Environmental Health. 2001. Technical report: mercury in the environment: implications for pediatricians. Pediatrics 108(1):197–205.

Grandjean P, Budtz-Jorgensen E, White RF, Jorgensen PJ, Weihe P, Debes F, et al. 1999. Methylmercury exposure biomarkers as indicators of neurotoxicity in children age 7 years. Am J Epidemiol 150:301–305.

Grandjean P, Murata K, Budtz-Jorgensen E, Weihe P. 2004. Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort. J Pediatr 144(2):169–176.

Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, et al. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol Teratol 19(6):417–428.

Guimaraes JRD, Ikingura J, Akagi H. 2000. Methyl mercury production and distribution in river water-sediment systems investigated through radiochemical techniques. Water Air Soil Pollut 124(1–2):113–124.

Harada Y. 1968. Congenital (or fetal) Minamata disease. In: Minamata Disease (Study Group of Minamata Disease, eds). Kumamato, Japan:Kumamato University, 93–118.

IOM (Institute of Medicine). 1981. Costs of Environment-Related Health Effects: A Plan for Continuing Study. Washington, DC:National Academy Press.

Innis SM. 1991. Essential fatty acids in growth and development. Prog Lipid Res 30:39–103.

Jacobson JL, Jacobson SW. 1996. Intellectual impairment in children exposed to polychlorinated biphenyls *in utero*. N Engl J Med 335:783–789.

Jorgensen EB, Debes F, Weihe P, Grandjean P. 2004. Adverse Mercury Effects in 7 Year-Old Children as Expressed as Loss in "IQ." Odense:University of Southern Denmark. Available: http://www.chef-project.dk/PDF/iq04louise5.pdf [accessed 15 May 2004].

Kjellstrom T, Kennedy P, Wallis S, Mantell C. 1986. Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage I: Preliminary Tests at Age 4. Report 3080. Solna, Sweden:National Swedish Environmental Protection Board.

Kjellstrom T, Kennedy P, Wallis S, Stewart A, Friberg L, Lind B, et al. 1989. Physical and Mental Development of Children with Prenatal Exposure to Mercury from Fish. Stage II: Interviews and Psychological Tests at Age 6. Report 3642. Solna, Sweden:National Swedish Environmental Protection Board.

Kris-Etherton PM, Harris WS, Appel LJ, for the Nutrition Committee. 2002. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. Circulation 106:2747–2757.

Landrigan PJ, Goldman L. 2003. Prenatal methylmercury exposure in the Seychelles [Letter]. Lancet 362(9384):666.

Landrigan PJ, Schechter CB, Lipton JM, Fahs MC, Schwartz J. 2002. Environmental pollutants and disease in American children: estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. Environ Health Perspect 110:721–728.

Larque E, Demmelmair H, Koletzko B. 2002. Perinatal supply and metabolism of long-chain polyunsaturated fatty acids: importance for the early development of the nervous system. Ann NY Acad Sci 967:299–310.

Leigh JP, Markowitz S, Fahs M, Shin C, Landrigan PJ. 1997. Costs of occupational injuries and illnesses. Arch Intern Med 157:1557–1568.

Lupton C, Burd L, Harwood R. 2004. Cost of fetal alcohol spectrum disorders. Am J Med Genet 127C(1):42–50.

Mahaffey KR, Clickner RP, Bodurow CC. 2004. Blood organic mercury and dietary mercury intake: National Health and Examination Survey, 1999 and 2000. Environ Health Perspect 112:562–570.

Mason RP, Reinfelder JR, Morel FMM. 1995. Bioaccumulation of mercury and methylmercury. Water Air Soil Pollut 80:915–921.

Max W, Rice DP, Sung H-Y, Michel M. 2002. Valuing Human Life: Estimating the Present Value of Lifetime Earnings, 2000. San Francisco:Institute for Health & Aging.

Murata K, Weihe P, Budtz-Jorgensen E, Jorgensen PJ, Grandjean P. 2004. Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury. J Pediatr 144(2):177–183.

Myers GJ, Davidson PW, Cox C, Shamlaye CF, Palumbo D, Cernichiari E, et al. 2003. Prenatal methylmercury exposure from the ocean fish consumption in the Seychelles child development study. Lancet 361:1686–1692.

National Marine Fisheries Service. 2004. Fisheries of the United States—2002. Silver Spring, MD:National Marine Fisheries Service, National Oceanic and Atmospheric Administration. Available: http://www.st.nmfs.gov/st1/fus/current/2002-fus.pdf [accessed 24 May 2004].

National Research Council. 2000. Toxicological Effects of Methylmercury. Washington, DC:National Academy Press.

Needleman HL, McFarland C, Ness RB, Fienberg SE, Tobin MJ. 2002. Bone lead levels in adjudicated delinquency: a case-control study. Neurotoxicol Teratol 24:711–717.

Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB. 1996. Bone lead levels and delinquent behavior. JAMA 275:363–369.

Neumann RM, Ward SM. 1999. Bioaccumulation and biomagnification of mercury in two warmwater fish communities. J Freshwater Ecol 14(4):487–498.

Nevin R. 2000. How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy. Environ Res 83:1–22.

Nriagu JO. 1989. A global assessment of natural sources of atmospheric trace metals. Nature 338:47–49.

Oken E, Kleinman KP, Berland WE, Simon SR, Rich-Edwards JW, Gillman MW. 2003. Decline in fish consumption among pregnant women after a national mercury advisory. Obstet Gynecol 102(2):346–351.

Opler MG, Brown AS, Graziano J, Desai M, Zheng W, Schaefer C, et al. 2004. Prenatal lead exposure, delta-aminolevulinic acid, and schizophrenia. Environ Health Perspect 112:548–552.

Rice DC, Schoeny R, Mahaffey K. 2003. Methods and rationale for a reference dose for methylmercury by the U.S. EPA. Risk Anal 23(1):107–115.

Rodier PM. 1995. Developing brain as a target of toxicity. Environ Health Perspect 103(suppl 6):S73–S76.

Salkever DS. 1995. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. Environ Res 70:1–6.

Schober SE, Sinks TH, Jones RL, Bolger PM, McDowell M, Osterloh J, et al. 2003. Blood mercury concentrations in US children and women of childbearing age, 1999–2000. JAMA 289:1667–1674.

Schwartz J. 1994. Societal benefits of reducing lead exposure. Environ Res 55:105–124.

Schwartz J, Pitcher H, Levin R, Ostro B, Nichols AL. 1985. Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis. EPA-230/05-85/006. Washington, DC:U.S. Environmental Protection Agency.

Seigneur C, Vijayaraghavan K, Lohman K, Karamchandani P, Scott C. 2004. Global source attributions for mercury deposition in the United States. Environ Sci Technol 38(2):555–569.

Smith KR, Corvalin CF, Kjellstrom T. 1999. How much global ill health is attributable to environmental factors? Epidemiology 10:573–584.

Stern AH. 2005. A revised probabilistic estimate of the maternal methyl mercury intake dose corresponding to a measured cord blood mercury concentration. Environ Health Perspect 113:155–163.

Stern AH, Smith AE. 2003. An assessment of the cord blood-maternal blood methylmercury ratio: implications for risk assessment. Environ Health Perspect 111:1465–1470.

Steuerwald U, Weihe P, Jorgensen PJ, Bjerve K, Brock J, Heinzow B, et al. 2000. Maternal seafood diet, methylmercury exposure and neonatal neurological function. J Pediatr 136:599–605.

Stretesky PB, Lynch MJ. 2001. The relationship between lead exposure and homicide. Pediatr Adolesc Med 155:579–582.

Szitanyi P, Koletzko B, Mydlilova A, Demmelhair H. 1999. Metabolism of ^{13}C-labeled linoleic acid in newborn infants during the first week of life. Pediatric Res 45:669–673.

Trasande L, Landrigan PJ. 2004. The National Children's Study: a critical national investment. Environ Health Perspect 112:A789–A790.

UNEP. 2002. Global Mercury Assessment Report. New York:United Nations Environmental Programme. Available: http://www.chem.unep.ch/mercury/Report/GMA-report-TOC.htm [accessed 17 May 2004].

U.S. Department of Energy, Office of Fossil Energy. 2004. Mercury Controls. Washington, DC:U.S. Department of Energy. Available: http://www.fossil.energy.gov/programs/powersystems/pollutioncontrols/overview_mercurycontrols.shtml [accessed 18 May 2004].

U.S. EPA. 1996. Mercury Study Report to Congress. Volume II: An Inventory of Anthropogenic Mercury Emissions in the United States. EPA452/R-96-001b. Washington, DC:U.S. Environmental Protection Agency.

U.S. EPA. 1997. Locating and Estimating Air Emissions from Sources of Mercury and Mercury Compounds. EPA-454/R-97-012. Research Triangle Park, NC:Office of Air Quality Planning and Standards and Office of Air and Radiation, U.S. Environmental Protection Agency.

U.S. EPA. 2003a. National Emissions Inventories for Hazardous Air Pollutants, 1999. Version 3, July 2003. Washington, DC:U.S. Environmental Protection Agency, Technology Transfer Network, Clearinghouse for Inventories and Emissions Factors. Available: http://www.epa.gov/ttn/chief [accessed 18 May 2004].

U.S. EPA. 2003b. National Toxics Inventory, 1990, Version 0302, October 2003. U.S. Environmental Protection Agency, Technology Transfer Network, Clearinghouse for Inventories and Emissions Factors. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/ttn/chief [accessed 18 May 2004].

U.S. EPA. 2004a. EPA Proposes Options for Significantly Reducing Mercury Emissions from Electric Utilities. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/air/mercuryrule/hg_factsheet1_29_04.pdf [accessed May 7, 2004].

U.S. EPA. 2004b. Clean Air Act. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/oar/oaq_caa.html [accessed 17 May 2004].

U.S. EPA. 2004c. Section B: Human and Environmental Benefits. 2002 Technical Support Package for Clear Skies. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/clearskies/tech_sectionb.pdf [accessed 17 May 2004].

U.S. EPA. 2004d. Mercury study report to Congress: Overview. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/mercury/reportover.htm [accessed 1 November 2004].

U.S. EPA. 2004e. Hospital/Medical/Infectious Waste Incinerators. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/ttnatw01/129/hmiwi/rihmiwi.html [accessed 18 May 2004].

U.S. EPA. 2004f. Rule and Implementation Information for Large Municipal Waste Combustors. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/ttn/atw/129/mwc/rimwc.html [accessed 18 May 2004].

U.S. EPA. 2004g. Rule and Implementation Information for Small Municipal Waste Combustors. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/ttn/atw/129/mwc/rimwc2.html [accessed 18 May 2004].

U.S. EPA. 2004h. Fish Advisories. Washington, DC:U.S. Environmental Protection Agency. Available: http://epa.gov/waterscience/fish/states.htm [accessed 9 November 2004].

U.S. EPA. 2005. Clean Air Mercury Rule: Basic Information. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/air/mercuryrule/basic.htm [accessed 1 April 2005].

Viscusi WK, Aldy JE. 2004. The Value of a Statistical Life: A Critical Review of Market Estimates throughout the World. NBER Working Paper Series, Working Paper 9487. Cambridge, MA:National Bureau of Economic Research. Available: http://www.nber.org/papers/w9487 [accessed 2 July 2004].

Wasserman GA, Liu X, Popovac D, Factor-Litvak P, Kline J, Waternaux C, et al. 2000. The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. Neurotoxicol Teratol 22(6):811–818.

Dove Chas l

ARTICLE IN PRESS

ELSEVIER

Health & Place ▮ (▮▮▮▮) ▮▮▮–▮▮▮

HEALTH & PLACE

www.elsevier.com/locate/healthplace

Environmental mercury release, special education rates, and autism disorder: an ecological study of Texas

Raymond F. Palmer[a,*], Steven Blanchard[b], Zachary Stein[a], David Mandell[c], Claudia Miller[a]

[a]*University of Texas Health Science Center, San Antonio Department of Family and Community Medicine, 7703 Floyd Curl Drive, San Antonio, Texas 78229-3900, USA*

[b]*Department of Sociology, Our Lady of the Lake University, San Antonio, Texas, USA*

[c]*University of Pennsylvania Center for Mental Health Policy and Services Research, USA*

Accepted 1 November 2004

Abstract

The association between environmentally released mercury, special education and autism rates in Texas was investigated using data from the Texas Education Department and the United States Environmental Protection Agency. A Poisson regression analysis adjusted for school district population size, economic and demographic factors was used. There was a significant increase in the rates of special education students and autism rates associated with increases in environmentally released mercury. On average, for each 1000 lb of environmentally released mercury, there was a 43% increase in the rate of special education services and a 61% increase in the rate of autism. The association between environmentally released mercury and special education rates were fully mediated by increased autism rates. This ecological study suggests the need for further research regarding the association between environmentally released mercury and developmental disorders such as autism. These results have implications for policy planning and cost analysis.

Keywords: Mercury; Special education; Autism; Environmental toxins; Ecological

Introduction

Exposure to a variety of environmental neurotoxicants is known to affect normal child development, resulting in a spectrum of adverse outcomes, ranging from severe mental retardation and developmental disability to more subtle changes in functioning, depending in part on the timing and dose of the chemical agent (Landrigan and Garg, 2002; Mendola et al., 2002; Rice and Barone, 2000).

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) section 104 (i), as amended by the Superfund Amendments and Reauthorization Act (SARA), requires the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA) to prepare a list, in order of priority, of substances that are most commonly found at waste facilities on the National Priorities List (NPL) and which are determined to pose the most significant potential threat to human health due to their known or suspected toxicity and potential for human exposure. Accordingly, mercury is listed as the third-most frequently found (arsenic and lead are

*Corresponding author. Tel.: +210 358 3883.
E-mail address: palmerr@uthscsa.edu (R.F. Palmer).

1353-8292/$ - see front matter © 2005 Published by Elsevier Ltd.
doi:10.1016/j.healthplace.2004.11.005

first and second) toxic substance in the United States (ATSDR, 2001).

Symptoms of nervous system disruption associated with chronic exposure to mercury has been known since the 19th century, when mercury was widely used in the felt industry which led to the expression of "hatter's disease" (Hu, 1998). Further epidemiological evidence of the neurotoxicity of mercury dates back to the 1950s, when it was ascertained that thousands of people in Minamata and Niigata Japan suffered various neurological impairments caused by consumption of mercury contaminated fish (Harada, 1978). However, the neurotoxicity of low-level mercury exposure has only recently been documented (NAS, 2000; EPA, 1997) and recent reports implicate mercury in the etiology of various developmental and learning disabilities (Ramirez et al., 2003; Grandjean et al., 2003) including autism (Bernard et al., 2001, 2002).

Recent evidence for mercury toxicity relevant to the biology of autism is compelling (Palomo et al., 2003; Aschner and Walker, 2002; Bernard et al., 2002; Vojdani et al., 2003) and Bradstreet et al. (2003) report that levels of urinary mercury after a 3-day treatment with an oral chelating agent, meso-2,3-dimercaptosuccinic acid (DMSA), in children with autistic spectrum disorders were three times those in a matched normal control sample.

Environmentally released mercury is a major source of mercury exposure. Mercury is released into the environment largely from fossil fuel (mainly coal) combustion by electrical utilities and from municipal and medical waste incinerators. This inorganic mercury becomes airborne and may be carried for miles before being deposited on soil or water. This inorganic form of mercury is then converted to a toxic form (methylmercury) by chemical reactions or by bacteria, which is absorbed by aquatic microorganisms that are eaten by fish, and in this manner accumulates up the aquatic food chain. Humans are primarily exposed through fish consumption (Myers et al., 2000) and transmission from mothers to infants is well documented in animal models (Newland et al., 1994) and human studies (Ramirez et al., 2000; Grandjean et al., 1995). Results from several studies show that maternal mercury exposure during pregnancy is associated with neuropsychological deficits in children and that this association is most evident in women with stable exposures throughout pregnancy (Ramirez et al., 2003; Grandjean et al., 2003).

Other than accidental poisoning at the population level, where developmental disabilities have been reported as the result of large mercury spills (Racz and Vandewater, 1982), there have been no published studies examining the risk of disability associated with mercury released into the environment within the current legal limits. The available information regarding exposure to toxic agents associated with developmental disorders is suggestive but inconclusive (Ostrowski et al., 2003). In a prior study, we report evidence for an association between environmentally released mercury and various developmental disorders, including autism, at the state level ($n = 50$) (unpublished manuscript). We considered the positive association between developmental disabilities and environmentally released mercury in that investigation as preliminary due to the relatively small number of large geological regions. In this study, we investigate the association between environmentally released mercury pollution and autism rates at the county ($n = 254$) and school district level ($n = 1184$) in Texas. The advantage of using county level data in this study allows an investigation using greater numbers of smaller geographic units in the analysis—this can potentially increase our power to detect an effect if in fact it present. Since Texas ranks 4th among states with the highest reported mercury releases (next to California, Oregon, and West Virginia) (USEPA-TRI, 2004), analysis of data from this state can be useful for further investigation of the association between environmental mercury release and developmental disorders. In this study, we investigate the association between total special education rates, autism, and environmental mercury release.

Methods

Data source and sample data regarding environmentally released mercury for each county were obtained from the United State Environmental Protection Agency Toxics Release Inventory (TRI) (USEPA-TRI, 2004). TRI collects information about chemical releases and waste management reported by major industrial facilities in the US. The TRI database was established by Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986 (EPCRA). Under EPCRA, industrial facilities in specific sectors are required to report their environmental releases and waste management practices annually to the EPA. Facilities covered by this act must disclose their releases to air, water, and land of approximately 650 toxic chemicals, as well as the quantities of chemicals they recycle, treat, burn, or otherwise dispose of on-site and off-site. The current analysis uses reports of pollution that industrial facilities provided to TRI for the calendar year 2001. The total number of pounds of environmentally released mercury was obtained for each county.

Administrative data from the Texas Education Agency (TEA) from school years 2000–2001 were analyzed. Data and data description are available at the TEA website at http://198.214.99.202. In compliance with the Texas Education Code, the Public Education Information Management System (PEIMS) contains data necessary for the legislature and the TEA to perform their legally authorized functions in overseeing public education. The database consists of student demographic, personnel, financial, and organizational information. Autism counts per school district were obtained by special request from the TEA. Data were from 1184 school districts in 254 counties in Texas. These districts represented approximately 4 million children enrolled in grades K through 12.

Diagnosis of autistic disorder was abstracted from the school record for each year of the study period. Diagnoses were made by qualified special education psychologists employed by the TEA or from psychologists or medical doctors outside the TEA system. While diagnoses were not standardized, there is considerable evidence that diagnoses of autistic disorder are made with good reliability and specificity in the field (Eisenmajer et al., 1996; Hill et al., 2001, Mahoney et al., 1998).

District population wealth was calculated as a school district's total taxable property value in 2001 as determined by the Comptroller's Property Tax Division (CPTD), divided by the total number of students in the district in 2000–2001. Property value was determined by the CPTD as part of its annual study, which attempts to present uniformly appraised property valuations statewide. The CPTD value is calculated by applying ratios created from uniform independent appraisals to the district's assessed valuations.

Racial composition was accounted for by the proportion of European-American children enrolled in schools within each district.

Total number of students was calculated as all enrolled students as of October 28, 2000 in grades kindergarten through twelve, who attended at least 1 day of school for that school year. Statewide, 6975 students, or 0.2% of all students, were enrolled but did not attend school.

Proportion of economically disadvantaged students was calculated as the percentage of students who were eligible for free meals under the National School Lunch and Child Nutrition Program, reduced-price meals under the National School Lunch and Child Nutrition Program, or other public assistance.

Total number of students enrolled in special education was calculated as the number of students receiving special education in each district.

Urbanicity. Eight separate demographic district regions were utilized in the analysis: (1) *Major urban* districts are the districts with the greatest membership in counties with populations of 650,000 or more, and more than 35% of the students are identified as economically disadvantaged. (2) *Other central city*—The major school districts in other large, but not major, Texas cities. Other central city districts are the largest districts in counties with populations between 100,000 and 650,000 and are not contiguous to any major urban districts. (3) *Major suburban* districts are contiguous to major urban districts. If the suburban district is not contiguous, it must have a student population that is at least 15% of the size of the district designated as major urban. (4) *Other central city suburban*—Other school districts in and around the other large, but not major, Texas cities. They are contiguous to other central city districts. If the suburban district is not contiguous, it must have a student population that is at least 15% of the size of the district designated as central city. (5) *Independent town*—The largest school districts in counties with populations of 25,000–100,000. (6) *Non-metro: fast growing* school districts that are not in any of the above categories and that exhibit a 5-year growth rate of at least 20%. These districts must have at least 300 students in membership. (7) *Non-metro: stable* school districts that are not in any of the above categories, yet have a number of students in membership that exceeds the state median. (8) *Rural* school districts that do not meet the criteria for placement into any of the above categories. These districts either have a growth rate less than 20% and the number of students in membership is between 300 and the state median, or the number of students in membership is less than 300.

In the analysis, the first two categories above were combined to form an "urban" dummy variable, categories three and four were combined to form a "suburban" dummy variable and categories five through seven formed an "other" category, with rural districts as the reference group.

Statistical methods. Since the 1184 school districts were nested within 254 counties, we modeled the data using a multilevel Poisson regression model to adjust estimates due to a potential county level clustering effect—which can bias estimated standard errors downward, thus leading to type I errors if not properly addressed (Barcikowski, 1981).

A multilevel Poisson regression model allowing for over-dispersion of the dependent variable was used in which the total number of children with autism and the number of special education students (excluding autism) was modeled separately as a function of the total pounds of environmentally released mercury. The model was adjusted for percent of the population of European-American descent, district population wealth, percent economically disadvantaged and urbanicity. Rates were offset by the total number of children served in a school district. For the model predicting autism rates, special education counts were included as a covariate in a subsequent model. For the model predicting special education rates, autism counts were also included as a covariate in a separate model. All models were estimated using MLwiN software with a log link function specified (Goldstein et al., 1998). The analysis yields adjusted relative rate estimates as a function of pounds of environmentally released mercury.

higher rate of autism in suburban relative to rural districts.

In model 2, after adjustment for the number of special education students, mercury remained a significant predictor of autism rates, indicating a 17% increase in autism rates for every 1000 lb of mercury released in the environment. The number of special education students was a significant predictor of autism rates as well. Wealth was no longer a significant predictor and the other covariates showed decreases relative to model 1, but remained significant.

Table 3 shows the regression estimates where special education rates (excluding autism counts) were modeled as a function of pounds of mercury and sociodemographic covariates (model 3), plus adjustment for the number of autistic students (model 4).

Model 3 shows that each 1000 lb of reported mercury release is associated with a 43% increase in the rate of special education students. Small but significant increases were associated with the percentage of European Americans, economically disadvantaged and district wealth. Community type was strongly associated with special education rates. All community-type categories show a much higher percentage of special education students relative to rural communities.

In model 4, after adjusting for total autism counts, the association between pounds of mercury and special education rates was no longer statistically significant—with the other covariates in the model remaining significant. This indicates that increased rates in autism account for the association between environmentally released mercury and the rate of special education students.

Discussion

To the best of our knowledge, this is one of the first investigations to report an ecological association between developmental disorders and environmentally released mercury.

The results of this study demonstrate that school district autism and special education rates are significantly associated with environmentally released mercury. This association was independent of the number of children served in the educational system for that district, district wealth, ethnic make-up, and community type. Further, these results indicate that the association between mercury release and school district special education rates was completely accounted for by increased rates of autism. This indicates that, in Texas, the increase in special education rates attributable to environmental mercury can be explained by increases in autism. The results of this study are consistent with our prior nation-wide study where an association between various developmental disabilities and environmentally released mercury was observed at the state level

Table 3
Poisson regression estimates predicting relative rate of special education prevalence

	Estimate (SE)	Relative rate	Lower 95% CI	Upper 95% CI
Model 3: Predicting special education prevalence rates as a function of mercury with demographic adjustments				
Mercury (per 1000 pounds)	0.360 (0.030)	1.433	1.350	1.522
Percent white	0.004 (0.001)	1.004	1.002	1.006
District wealth (per $100,000)	0.050 (0.010)	1.051	1.030	1.073
Percent economically disadvantaged	0.012 (0.001)	1.012	1.010	1.014
Urban versus rural	2.741 (0.104)	15.502	12.591	19.087
Suburban versus rural	2.110 (0.103)	8.248	6.713	10.135
Other versus rural	1.550 (0.110)	4.711	3.781	5.871
Model 4 Predicting special education prevalence rates as a function of mercury with demographic and autism count adjustments				
Mercury (per 1000 pounds)	−0.062 (0.032)	0.940	0.882	1.002
Percent white	0.008 (0.001)	1.008	1.006	1.010
District wealth (per $100,000)	0.030 (0.010)	1.030	1.010	1.051
Percent economically disadvantaged	0.014 (0.001)	1.014	1.012	1.016
Urban versus rural	2.240 (0.068)	9.393	8.199	10.762
Suburban versus rural	1.902 (0.066)	6.699	5.871	7.645
Other versus rural	1.174 (0.073)	3.235	2.795	3.743
Autism count (per 100)	0.689 (0.022)	1.992	1.906	2.081

Results

Table 1 shows the descriptive statistics of the study variables. The standard deviation and the maximum and minimum values indicate considerable variation for all study variables. Table 2 shows the results of the regression model where autism rates were modeled as a function of pounds of mercury and sociodemographic covariates (model 1), plus adjustment for the number of special education students (excluding autism) (model 2).

Model 1 shows that for each 1000 lb of environmentally released mercury, the rate of autism increases by 61%. A small but significant rate increase is noted for districts with higher wealth, and a small but significant inverse association is observed for percentage of European American and economically disadvantaged students. A large effect is observed for community type. The highest rate increase is observed when comparing urban to rural school districts—relative to rural districts there is a 473% higher rate of autism. There is a 255%

Table 1
Descriptive statistics for study variables ($n = 1184$ school districts in 254 counties)

	Mean	SD	Minimum	Maximum
Autism count total	5.11	21.39	0	416
Total special education population count	414.12	1205.21	0	21,900
Pounds of environmental mercury release	203.99	522.84	0	2059
Total student population	3382.30	10908.99	6	209,916
Percent economically disadvantaged	47.28	21.70	0	100
Percent European American	58.33	29.71	0	100
District wealth	$189,080	$262,290	0	$4,276,736
Community type				
% Urban	4.1	—	—	—
% Suburban	13.2	—	—	—
% Rural	34.9	—	—	—
% Other	47.8	—	—	—

Table 2
Poisson regression estimates predicting relative rate of autism prevalence

	Estimate (SE)	Relative rate	Lower 95% CI	Upper 95% CI
Model 1: Predicting autism prevalence rates as a function of mercury release with demographic covariate adjustments				
Mercury (per 1000 pounds)	0.479 (0.041)	1.614	1.487	1.752
Percent European American	−0.023 (.001)	0.977	0.975	0.979
District wealth (per 100,000 dollars)	0.060 (0.010)	1.062	1.041	1.083
Percent economically disadvantaged	−0.029 (0.001)	0.971	0.969	0.973
Urban versus rural	1.553 (0.109)	4.726	3.800	5.877
Suburban versus rural	0.935 (0.108)	2.547	2.052	3.161
Other versus rural	0.027 (0.112)	1.027	0.821	1.285
Model 2: Predicting autism prevalence rates as a function of mercury with demographic and special education count adjustment				
Mercury (per 1000 pounds)	0.160 (0.031)	1.174	1.103	1.249
Percent European American	−0.019 (0.001)	0.981	0.979	0.983
District wealth (per 100,000 dollars)	0.010 (0.010)	1.010	0.990	1.030
Percent economically disadvantaged	−0.034 (0.001)	0.967	0.965	0.969
Urban versus rural	0.953 (0.078)	2.593	2.219	3.031
Suburban versus rural	0.808 (0.074)	2.243	1.935	2.601
Other versus rural	−0.356 (0.087)	0.700	0.589	0.834
Special education count (per 1000)	0.172 (0.005)	1.188	1.176	1.200

(unpublished manuscript). However, the results of this report should be interpreted with caution for a number of reasons.

First, this is an ecological study that precludes interpretation at the individual level. We have used aggregate units in this analysis to investigate differential rates of autism as a function of pounds of mercury at the county level. While we properly addressed the potentially biasing effects of clustering (school districts nested within counties) by utilizing appropriate analytic methods (e.g. multilevel-analysis), individual data are required to make a better case for the observed associations and their interpretations. Nevertheless, ecological studies of this type are often an important first step in identifying subsequent areas of investigation.

Second, a causal association between environmentally released mercury and developmental disorders cannot be determined from this cross-sectional data. Data availability permitting, future studies could investigate this association by using longitudinal data where changes in mercury levels over time may be used as a predictor of the rate of change in developmental disorders over time.

Third, we should consider that school-based administrative autism data, such as these, are only a proxy for true community prevalence. However, these autism rates are most likely biased downward. For example, Yeargin-Allsopp et al. (2003) found that, in one metropolitan area, 18% of children who qualified for a diagnosis of autism according to their study criteria were receiving special education services but had not been categorized as having autism. The critical unknown issue is whether identification of children in the special education system is systematically biased in the same direction as reporting of environmental mercury release. For example, counties in which administrations are more aggressive regarding penalties for underreporting toxic release may also have educational policies that result in a greater number of children identified for special education services. Despite the limitations of these administrative data, as demonstrated, these data can be a useful component to preliminary epidemiological studies (Dales et al., 2001). By demonstrating an association between environmentally released mercury and developmental disorders, the results of this study provide a necessary first step in identifying plausible contributing factors of risk for developmental disabilities.

This line of research has implications for toxic substance regulation and prevention policies. The effects of differing state policies regarding toxic release of mercury on the incidence of developmental disorders should be investigated. For example, policies that have successfully limited exposures to lead have had direct effects on morbidity and have demonstrated reductions in health care costs related to lead exposure (Sargent et al., 1999, Galke et al., 2001; Brown, 2002). However, while federal efforts toward reducing mercury exposure through policy have been successful to some extent by signing bills into law, proportionally few have been enacted (Mercury Policy Project (MMP), 2004). Despite existing policy recommendations, debate concerning acceptable levels of safety still remains (Dourson et al., 2001; Kaiser, 2000), thus, limiting progress toward evaluating policies related to reducing exposure to mercury.

Conclusions

What is currently known about the low-level toxicity of mercury from behavioral toxicology and behavioral teratology studies are convincing enough to warrant further study. This study is among the first to demonstrate an association between environmentally released mercury at the county level and the rate of developmental disability. Given the limitations of this ecological association, future studies should investigate this association using other methodologies and samples. This line of research has important implications for public health policy and supports prior recommendations for reducing environmentally released mercury (Needleman, 1995; Landrigan et al., 1994).

References

Agency for Toxic Substances and Disease Registry (ATSDR), 2001. CERCLA Priority List of Hazardous Substances. US Department of Health and Human Services, Public Health Service, Atlanta, GA. www.atsdr.cdc.gov/clist.html.

Aschner, M., Walker, S.J., 2002. The neuropathogenesis of mercury toxicity. Molecular Psychiatry 7 (Suppl. 2), S40–S41.

Barcikowski, R., 1981. Statistical power with group mean as the unit of analysis. Journal of Educational Statistics 6, 267–285.

Bernard, S., Enayati, A., Redwood, L., Roger, H., Binstock, T., 2001. Autism: a novel form of mercury poisoning. Medical Hypotheses 56, 462–471.

Bernard, S., Enayati, A., Roger, H., Binstock, T., Redwood, L., 2002. The role of mercury in the pathogenesis of autism. Molecular Psychiatry 7, S42–S43.

Bradstreet, J., Geier, D., Kartzinel, J., Adams, J., Geier, M., 2003. A case-control study of mercury burden in children with autistic spectrum disorders. Journal of American Physicians and Surgeons 8 (3), 76–79.

Brown, M.J., 2002. Costs and benefits of enforcing housing policies to prevent childhood lead poisoning. Medical Decision Making 22 (6), 482–492.

Dales, L., Hammer, S., Smith, N., 2001. Time trends in autism and in MMR immunization coverage in California. Journal of the American Medical Association 285 (9), 1183–1185.

Dourson, M.L., Wullenweber, A.E., Poirier, K.A., 2001. Uncertainties in the reference dose for methylmercury. Neurotoxicology 22 (5), 677–689.

Eisenmajer, R., Prior, M., Leekam, S., Wing, L., Gould, J., Welham, M., 1996. Comparison of clinical symptoms in autism and Asperger's disorder. Journal of the American Academy of Child & Adolescent Psychiatry 35 (11), 1523–1531.

Environmental Protection Agency, 1997. Mercury Study Report to congress, vol 1. Available at: http://www.epa.-gov/ttnatw01/112nmerc/volume1.pdf. Accessed February 22, 2004.

Galke, W., Clark, S., Wilson, J., et al., 2001. Evaluation of the HUD lead hazard control grant program: early overall findings. Environmental Research 86 (2), 149–156.

Goldstein, H., Rasbash, J., Plewis, I., Draper, D., Browne, W., Yang, M., Woodhouse, G., Healy, M.A., 1998. User's guide to MLwiN, Version I.0, January. Institute of Education. ISBN 085473 547X.

Grandjean, P., Weihe, P., White, R.F., 1995. Milestone development in infants exposed to methylmercury from human milk. Neurotoxicology 16, 27–33.

Grandjean, P., White, R.F., Weihe, P., Jorgensen, P.J., 2003. Neurotoxic risk caused by stable and variable exposure to methylmercury from seafood. Ambulatory Pediatrics 3 (1), 18–23.

Harada, M., 1978. Congenital Minamata disease: intrauterine methylmercury poisoning. Teratology 18, 285–288.

Hill, A., Bolte, S., Petrova, G., Beltcheva, D., Tacheva, S., Poustka, F., 2001. Stability and interpersonal agreement of the interview-based diagnosis of autism. Psychopathology 34 (4), 187–191.

Hu, H., 1998. Heavy metal poisoning. In: Fauci, A.S., Braunwald, E., Isselbacher, K.J., Wilson, J.D., Martin, J.B., Kasper, D.L., Hauser, S.L., Longo, D.L. (Eds.), Harrison's Principles of Internal Medicine, 14th ed. McGraw-Hill, New York, pp. 2564–2569 (Chapter 397).

Kaiser, J., 2000. Mercury report backs strict rules. Science 289, 371–372.

Landrigan, P.J., Garg, A., 2002. Chronic effects of toxic environmental exposures on children's health. Journal of Toxicology—Clinical Toxicology 40 (4), 449–456.

Landrigan, P.J., Graham, D.G., Thomas, R.D., 1994. Environmental neurotoxic illness: research for prevention. Environmental Health Perspectives 102 (Suppl. 2), 117–120.

Mahoney, W., Szatmari, P., MacLean, J., Bryson, S., Bartolucci, G., Walter, S., 1998. Reliability and accuracy of differentiating pervasive developmental disorder subtypes. Journal of the American Academy of Child & Adolescent Psychiatry 37 (3), 278–285.

Mendola, P., Selevan, S.G., Gutter, S., Rice, D., 2002. Environmental factors associated with a spectrum of neurodevelopmental deficits. Mental Retardation Developmental Disabilities Research Reviews 8 (3), 188–197.

Mercury Policy Project (MMP), 2004. Web site. Available at http://www.mercurypolicy.org. Accessed March 1.

Myers, G.J., Davidson, P.W., Cox, C., Shamlaye, C., Cernichiari, E., Clarkson, T.W., 2000. Twenty-seven years studying the human neurotoxicity of methylmercury exposure. Environmental Research 83 (3), 275–285.

National Academy of Sciences, 2000. Toxicological Effects of Methylmercury. National Academy Press, Washington, DC.

Needleman, H.L., 1995. Behavioral toxicology. Environmental Health Perspectives 103 (Suppl. 6), 77–79.

Newland, M.C., Yezhou, S., Logdberg, B., Berlin, M., 1994. Prolonged behavioral effects of in utero exposure to lead or methyl mercury: reduced sensitivity to changes in reinforcement contingencies during behavioral transitions and in steady state. Toxicology and Applied Pharmacology 126, 6–15.

Ostrowski, S., Wilbur, S., Chou, C., Pohl, H., Stevens, Y., Allred, P., Roney, N., Fay, M., Tylenda, C., 2003. Agency for Toxic Substances and Disease Registry's 1997 priority list of hazardous substances. Latent effects—carcinogenesis, neurotoxicology, and developmental deficits in humans and animals. Toxicology & Industrial Health 15 (7), 602–644.

Palomo, T., Beninger, R.J., Kostrzewa, R.M., Archer, T., 2003. Brain sites of movement disorder: genetic and environmental agents in neurodevelopmental perturbations. Neurotoxological Research 5 (1–2), 1–26.

Racz, W., Vandewater, L., 1982. Perspectives on the central nervous system toxicity of methylmercury. Canadian Journal of Physiology and Pharmacology 60, 1037–1045.

Ramirez, G., Vince Cruz, C., Pagulayan, O., Ostrea, E., Dalisay, C., 2000. The Tagum Study I: analysis and clinical correlates of mercury in maternal and cord blood, breast milk, meconium, and infants' hair. Pediatrics 106 (4), 774–781.

Ramirez, G., Pagulayan, O., Akagi, H., et al., 2003. Tagum study II: follow-up study at two years of age after prenatal exposure to mercury. Pediatrics 111 (3), e289–e295.

Rice, D., Barone, S., 2000. Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. Environmental Health Perspectives 108 (Suppl 3), 511–533.

Sargent, J.D., Dalton, M., Demidenko, E., Simon, P., Klein, R.Z., 1999. The association between state housing policy and lead poisoning in children. American Journal of Public Health 89 (11), 1690–1965.

United States Environmental Protection Agency Toxics Release Inventory, 2004. http://www.epa.gov/tri/. Accessed May.

USEPA-TRI, 2004.

Vojdani, A., Pangborn, J.B., Vojdani, E., Cooper, E.L., 2003. Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism. International Journal of Immunopathology and Pharmacology 16 (3), 189–199.

Yeargin-Allsopp, M., Rice, C., Karapurkar, T., Doernberg, N., Boyle, C., Murphy, C., 2003. Prevalence of autism in a US metropolitan area. Journal of the American Medical Association 289, 49–55.

ELSEVIER

Available online at www.sciencedirect.com

SCIENCE @ DIRECT®

Science of the Total Environment xx (2005) xxx–xxx

Science of the Total Environment

An International Journal for Scientific Research into the Environment and its Relationship with Humankind

www.elsevier.com/locate/scitotenv

Environmental costs of mercury pollution

Lars D. Hylander [a,*], Michael E. Goodsite [b,c,d,1]

[a] *Department of Earth Sciences, Air and Water Science, Uppsala University, Villavägen 16, S-752 36 Uppsala, Sweden*

[b] *Department of Chemistry, Environmental Chemistry Research Group, University of Southern Denmark, Campusvej 55, DK-5230 Odense M, Denmark*

[c] *Institute for Environmental geochemistry, University of Heidelberg, Im Neuenheimer Feld 236, 69120 Heidelberg, Germany*

[d] *Department of Chemistry, University of Pennsylvania, 231 S. 34th St., Philadelphia, PA 19104-6323, USA*

Received 2 November 2004; received in revised form 3 November 2005; accepted 29 November 2005

Abstract

Mercury (Hg) has been used for millennia in many applications, primarily in artisanal mining and as an electrode in the chlor–alkali industry. It is anthropogenically emitted as a pollutant from coal fired power plants and naturally emitted, primarily from volcanoes. Its unique chemical characteristics enable global atmospheric transport and it is deposited after various processes, ultimately ending up in one of its final sinks, such as incorporated into deep sediment or bioaccumulated, primarily in the marine environment. All forms of Hg have been established as toxic, and there have been no noted biological benefits from the metal. Throughout time, there have been notable incidents of Hg intoxication documented, and the negative health effects have been documented to those chronically or acutely exposed. Today, exposure to Hg is largely diet or occupationally dependent, however, many are exposed to Hg from their amalgam fillings. This paper puts a tentative monetary value on Hg polluted food sources in the Arctic, where local, significant pollution sources are limited, and relates this to costs for strategies avoiding Hg pollution and to remediation costs of contaminated sites in Sweden and Japan. The case studies are compiled to help policy makers and the public to evaluate whether the benefits to the global environment from banning Hg and limiting its initial emission outweigh the benefits from its continued use or lack of control of Hg emissions. The cases we studied are relevant for point pollution sources globally and their remediation costs ranged between 2500 and 1.1 million US$ kg^{-1} Hg isolated from the biosphere. Therefore, regulations discontinuing mercury uses combined with extensive flue gas cleaning for all power plants and waste incinerators is cost effective.

Keywords: Arctic; Contamination; Economic; Japan; Prevent pollution; Remediation; Sweden

1. Introduction

Since ancient times, man has been aware of the toxic nature of elemental mercury (Hg) and since, its salts and organic compounds. Most are familiar with Hg intoxication, having heard of "mad hatters" hat makers who used fur treated with mercuric nitrate. In industrialized nations, health concern was, until a few decades ago, focused on occupational exposure, e.g. in mines, even though there were several documented incidents of local Hg intoxication within the past 50 years and evidence of wildlife mortality in the 1960s. This lead the Swedes and Finns to initiate studies on the risks of using Hg, where knowledge was acquired about environmental transformations of inorganic Hg to far more toxic organic forms along, initially, largely unknown transfer pathways with emissions from the large quantities of Hg used in paper and chlor–alkali plants. (Hylander and Meili, 2005 and references therein).

In general, the state of Hg research is relatively immature compared with other trace metals, especially lead. Elemental Hg is unique when compared to other trace metals found in the atmosphere, in that it is approximately 95% in the gaseous elemental form (Slemr et al., 1985; Schroeder and Munthe, 1998), where other metals, e.g. lead, are primarily associated in the atmosphere as aerosols. The characteristics of gaseous metallic Hg, such as low aqueous solubility, mean that it has relatively low reactivity and is stable. Therefore, gaseous elemental mercury has a long atmospheric residence time, enabling global transport. Its vapor pressure allows it to be

* Corresponding author. Tel.: +46 18 471 22 65; fax: +46 18 55 11 24.
E-mail addresses: Lars.Hylander@hyd.uu.se (L.D. Hylander), meg@chem.sdu.dk (M.E. Goodsite).

[1] Presently visiting associate professor (February 2005 to February 2006) at the University of Heidelberg; visiting scientist (January 2004 to January 2005) at the University of Pennsylvania.

0048-9697/$ - see front matter © 2005 Elsevier B.V. All rights reserved.
doi:10.1016/j.scitotenv.2005.11.029

deposited and re-emitted, as does bacteriologic conversions and subsequent emission. All of these factors contribute to its spread throughout the globe to areas where there are very little natural or local man-made inputs, such as the Arctic. This global transport means that all people and environments will pay some type of cost as a Hg receptor, some more than others, due to enhanced deposition, as is the case in the Arctic. Characterizing this cost is a challenge that needs to be met to properly enact legislation regulating production, consumption and emission.

Although illegal pollution occurs, especially in countries with weak administrative and executive capacity and by persons with few moral restrictions, most pollution is legal in the aspect that emitters are generally meeting the national, legal requirements with respect to emission-control. However, the legal requirements are often far from sufficient to safeguard clean production processes and environmentally benign products. One of the reasons the legal requirements are lacking is that damages to the environment are often difficult to quantify, and even more difficult to assign a monetary value. In addition, there is a time lag between emission and effects. As a result, the environment has become a sink for Hg pollution. This is costly to society and causes a variety of costs, so-called externalities, not included in the prices the producers and consumers pay for products and services. Examples are reduced recreational value of polluted areas, culture losses, reduced biodiversity, fish and other food and feed resources not suitable for consumption or become extinct, and health effects from Hg entering the body via contaminated food, soil, water, and air.

Views on the issue of mercury are often politically charged, partially due to the costs involved in emission reduction. The views range from the far right, disregarding the need to reduce Hg emissions (e.g. Kava et al., 2004) to the far left, claiming that Hg must be banned immediately without any exception. In an effort to give policy makers and the public an objective, environmental, economic review of costs associated with mercury emission control and remediation, we have compiled data from several case studies. Our objectives are to identify and quantify environmental costs of Hg pollution and where possible, assign monetary values on Hg pollution. The aim is to present data possible to use in a global perspective by quantifying remediation costs for different case studies and compare these costs with preventive measures and potential effects in the Arctic, having hardly any anthropogenic Hg emission sources. The goal is to provide input to lawmakers and citizens with respect to Hg and its regulation and control. Since Hg has no redeeming biological use, all anthropogenic use and release is considered contaminant.

2. Material and methods

Costs

Environmental costs can in a general sense be defined as costs to the environment and health not included in the prices the producers and consumers pay for products and services. The costs may be divided into two types of categories: i) damage (or value-loss) costs, such as contaminated fish, and ii) policy costs incurred in responding to pollution damage plus the costs of changing practices to meet legal obligations (Pretty et al., 2003). Policy costs needed in responding to pollution damage but still not paid, because damage has still not been remediated, are occasionally termed environmental debt (Jernelöv, 1992). Total environmental costs comprise a wide range of costs, estimated according to different methodologies. Our aim is not to evaluate different methods, but to focus on certain costs caused by Hg pollution, which will be described briefly together with methods for cost calculation used.

Damage costs of commercially exploited resources such as fishery and organized tourism can be based on damage occurred and the actual market value. Damages to ecosystem services, public health, etc. are more difficult to valuate, partly because we know too little about the importance of ecosystem services and health effects from Hg, alone as well as in combination with other substances. Often, these costs are based on willingness to pay to avoid damage or willingness to accept compensation to tolerate e.g. a deteriorated health, although the latter choice is often not made before the damage has occurred. Policy costs may be based on estimated, or actual, whenever available, costs for remediation, prevention, and control.

We have restricted this study to policy costs for remediation at six industrial sites, to costs for preventing and controlling Hg emissions, and to food damage cost for the Arctic and potential loss of IQ in the Greenland population. To accomplish this, we reviewed literature on global Hg use, emissions, and pollution effects and studied remediation costs of different case studies and costs for measures to avoid mercury pollution. The absence of basic, quantified data on environmental effects from Hg pollution on a global scale as well as in the Arctic, caused that we could calculate a tentative damage cost only to food in the Arctic, excluding potential effects on biodiversity and most aspects of health, cultural loss and other aspects of the total damage costs.

The only health cost calculated here is the one due to loss of intelligence caused by excessive prenatal exposure to methyl Hg. The National Research Council (2000), based on studies in the Faroe Islands and New Zealand, concludes that developmental effects become apparent at levels around 5.8 μg Hg L^{-1} cord blood of newborn children (Kjellström, 1989; Grandjean et al., 1997). Above this limit, a linear relationship has been assumed with a loss of 1.5 IQ points for each doubling in cord blood Hg concentration (Trasande et al., 2005). Loss of one IQ point was calculated to correspond to a 2.578% decrease in lifetime expected earnings (average for males and females; Trasande et al., 2005). The value of lifetime earnings discounted to present value used in this study is 897 735 US$, which is the average for an American child born in 2000 with an assumed 1% annual growth in productivity and a 3% real discount rate as calculated by Max et al. (2000). The annual average number of births in Greenland for 1998–1999 was obtained from United Nations (UN, 2002). Mercury levels in cord blood were obtained from Bjerregaard and Hansen (2000), sampling 178 newborn children and their mothers in western Greenland, where most Greenlanders are living and where the Hg levels in humans are lower than in eastern and northern Greenland (Hansen et al., 1983; Hansen and Pedersen, 1986). Since no table was presented including all original data, we constructed a normal curve. As a conservative approach, this was based on the geometric mean, being lower than the arithmetic mean.

The remediation cases we present are mainly from Sweden, where about a dozen remediation projects involving Hg polluted sites have been or are being executed. Economic aspects of remediation at Minamata, Japan, the site of the most serious industry pollution from Hg, are also included.

Options to avoid emissions into the environment may, according to engineers' definition, be divided into three categories: a) preventive measures, b) primary control measures, and c) secondary control measures. Preventive measures are undertaken to prevent the generation of emissions. This could be fuel substitution and/or fuel washing with regard to the Hg emissions from power plants. At primary control measures, Hg emissions are generated but reduced at the moment of their generation, e.g. selection of various types of industrial technologies with lower combustion temperatures to reduce Hg emissions from power plants. At secondary control measures, Hg emissions are generated but removed later on from exhaust gases by flue gas desulfurization, application of electrostatic precipitators (ESP), carbon beds, etc. Here we present preventive measures and secondary control measures, because primary control measures are less efficient for Hg due to its characteristics such as low melting and boiling temperatures and high vapor pressure.

All monetary values are given in US$ by converting values in Swedish crowns by the monthly average exchange rate as of September 2004 (1 US$=7.4484 SEK) based on daily fix [(bid+ask)/2] (Riksbanken, 2004). Current prices have been used if otherwise not stated. In order to permit comparison of remediation costs between different case studies, the remediation cost per kilogram Hg recovered has been calculated. In case recovery of other toxics has been a contributing reason to carry out a remediation, the remediation costs should ideally be divided between the toxics. This partition is not evident, therefore, in those cases we present a figure where all remediation costs have been assigned Hg and mention types and quantities of other toxics secured.

3. Results and discussion

3.1. The Arctic

One of the most actual present day large-scale Hg problem is faced in the Arctic, where ecosystems and local communities are fragile due to high exposure, both from enhanced deposition as explained below, and dietary consumption, which may be synergistically multiplied by other organic pollutants bioaccumulated in the indigenous dietary sources (Grandjean et al., 2003). The Arctic as a region has no known significant natural or man-made sources of Hg, except for mines and smelters at the Kola and Taymyr peninsulas in Russia, annually emitting up to 5.5 metric tons Hg, including emissions from waste incineration and coal combustion in adjacent cities and gold mining in northeastern Siberia (calculated from ACAP, 2005). However, Hg's toxicity has already had measurable effects in the sub Arctic Faroe Islands (Grandjean et al., 1992, 1995, 1997; Sørensen et al., 1999; Steuerwald et al., 2000), which through its isolation and traditional marine diet, may be compared with other indigenous communities of the far North.

Schroeder et al. (1998) reported on their 1995 discovery of the springtime depletion of tropospheric, gaseous Hg in the high Canadian Arctic, leading to a greater than expected deposition of Hg to the Arctic. It has been dubbed atmospheric Hg depletion episodes, AMDE (Schroeder et al., 2003). Skov et al. (2004) predict that this perennial phenomenon results in that, over the entire Arctic region, approximately 200 metric tons Hg is annually deposited, most of it of anthropogenic origin. This is more than double the amount of Hg earlier thought to be deposited to the Arctic. The USA has the largest anthropogenic Hg emissions of the countries with part of the territory in the Arctic, based on both total and per capita emissions (Norwegian Pollution Control Authority, 2005). Globally, China is the dominating Hg polluter and a considerable part of this Hg together with large Hg emissions in Central and South Europe enters the Arctic (Pacyna and Keeler, 1995; Dastoor and Larocque, 2004).

With the current market price of liquid Hg at approximately 14 500 US$ per metric ton (Hayes, 2004), an oversimplified calculation shows that the price for these 200 tons of Hg, if sold on the market, would have been approximately 2.9 million US$. This is much less than the 2000 million US$ or more to salvage this amount of Hg from coal emissions (Poulson, 1994), which in this aspect appear to not be a cost effective measure.

3.1.1. Damage costs in the Arctic

Another aspect is to look at damages caused by these Hg emissions and related costs. There are no data on quantities of fish and other food, such as marine mammals, rejected in the Arctic because of Hg levels above a limit safe for consumption, but at continued emissions, an increasing quantity of fish and mammals caught will exceed this limit. The WHO guide line of 0.5 mg total Hg kg^{-1} f.w. is generally used as a limit (Galvão and Corey, 1987), although Canada (for those who consume large amounts of fish), China and Japan has lower limits (0.2–0.4 mg total Hg kg^{-1} f.w.; UNEP, 2002). Some other countries have a higher limit, 1.0 mg total Hg kg^{-1} f.w., for piscivorous fish species, a value based on commercial considerations and not on health aspects (UNEP, 2002). However, the 0.5-mg value should be halved to harmonize with the revised provisional tolerable weekly intake (PTWI) for methyl Hg, being reduced from 3.3 to 1.6 μg/kg body weight per week (UN-FAO/WHO-JECFA, 2003). This revision was made by the Joint FAO/WHO Expert Committee on Food Additives at their meeting in June 2003 and it was realized to sufficiently protect the developing fetus, exposed to methyl Hg through contaminated food eaten by the pregnant mother. In areas such as the Arctic, where the daily fish servings exceed 90 g day^{-1}, the safe fish Hg limit is lower than in other areas, resulting in that an important part of the catch is presently unsuitable for consumption (Johansen et al., 2004). Mercury content consistently exceeds guideline limits for subsistence consumption or commercial sale of lake trout and northern pike in the Canadian Shield lakes of the Northwest Territories and northern Quebec, and also burbot liver from Canadian Arctic, generally, exceed the limit (Braune et al., 1999).

Greenland, with most of its territory in the geographic area of the Arctic, has in average for the last 28-year period (1976–2003) landed 120 000 tons of fish and shellfish, which together with marine mammals captured, have resulted in the production of close to 58 000 tons year^{-1} of fish, shellfish and products from marine mammals (FAO, 2004). The median export price for the exported quantities in current values has been 0.95 US$ kg^{-1}, with the minimum value of 0.44 in 2001 and the maximum value of 1.89 in 1994. The median import price for fish and related products to Greenland has been more than twice as high at 2.30 US$ kg^{-1}, with the minimum value of 0.46 in 1984 and the maximum value of 4.17 in 1998. The value of the annual marine production is 54.8 million US$, if valued to the Greenland median export price, and 133.4 million US$, if valued to the median import price (FAO, 2004). The export price is motivated to use when valuating marine products exported, making up 6.0% of total marine production in Greenland (FAO, 2004). However, to valuate the domestic consumption, we have chosen to use the import price, because fish and mammals caught with excessive levels of pollutants have, in general, to be replaced with imports and not by reduced exports, because of the marginal quantities exported (6%), Based on this, the value of the average annual marine production in Greenland is 128.7 million US$. We have estimated the cost of Hg polluted marine products to be 24.5% of this value, corresponding to 31.5 million US$ year^{-1}. This fraction is based on that the Inuits of western Greenland would need to avoid certain components of their traditional food to not exceed health guidelines for pollutants. These food items make up 24–25% of their present diet and by excluding them, their Hg intake would decrease by 44% (Johansen et al., 2004).

In addition to Greenland, more than 20 other countries, having no or a limited part of their territory in the Arctic, are annually capturing about two million (2 074 829) tons fish and shellfish in Arctic waters to an annual value of 1000 million US$ (obtained by multiplying Greenland median export price with annual average captures for the last 28 years from the geographic Arctic, adjusted for losses from capture to sellable products with the same factor as for marine production in Greenland; data from FAO, 2004). This value is in parity with the cost of flue gas cleaning for anthropogenic Hg reaching the Arctic. Although not all fisheries in the Arctic are acutely threatened by Hg pollution, Inuits of eastern Canadian Arctic have a mean mercury intake calculated to be 122 μg day^{-1} for women and 166 μg day^{-1} for men (Zauke et al., 1994; Johansen et al., 2004). Since most of this Hg is in the methyl form, the intake is 5–7 times the revised, tolerable daily intake (UN-FAO/WHO-JECFA, 2003).

In North Greenland, where the highest Hg exposure in Greenlanders is documented, more than 80% of the population exceed the benchmark level of concern for the United States, 58 μg Hg L^{-1} blood, and 16% exceed the World Health Organization (WHO) minimum toxic blood concentration in non-pregnant adults, 200 μg Hg L^{-1} blood (Hansen and Pedersen, 1986). Weihe et al. (2002) suggest that observed neurobehavioral deficits in Inuit children from Qaanaaq, NW Greenland, might be related to dietary Hg exposure. Eating habits changing away from the traditional food, partly spurred by consumption advisories, has resulted in a reduced Hg burden of Inuit body tissues since the 1970's and 1980's (Hansen et al., 1983; Tulinius, 1995; Oostdam et al., 1999). Still, hair total Hg concentrations are often an order of magnitude higher nowadays among Inuts than the hair Hg concentrations among inhabitants of sub Arctic Alaska before industrialization (Egeland et al., 1999). Present hair methyl Hg concentrations may diverge even more than total Hg from pre-industrial concentrations (Egeland et al., 1999; Rothschild and Duffy, 2002).

Altogether, this indicates that many Inuits are exposed to alarmingly high prenatal and postnatal exposure to methyl Hg. Cord blood Hg levels above 5.8 μg L^{-1} of born children are associated with loss of intelligence, which causes diminished economic productivity that persists over the entire lifetime of these children (Kjellström, 1989; Grandjean et al., 1997; National Research Council, 2000). This cost of methyl mercury toxicity is calculated to 8700 million US$ annually (range, 2200–43 800 million US$ in 2000 US$) in the USA alone (Trasande et al., 2005).

Nearly 3/4 of the children born in Greenland are estimated to have cord blood Hg levels above 5.8 μg L^{-1} (Table 1). Thereby the share of children with neurodevelopment deficiencies caused by methyl Hg is markedly larger in Greenland than in the USA, assuming that the sensitivity to methyl Hg damage is comparable in the two populations. However, the cost related to the damage is smaller in Greenland, because of fewer inhabitants there. Nevertheless, the cost is important to calculate and should be added to national figures of costs related to Hg in countries with anthropogenic Hg emissions. Based on data and calculations as presented in the Material and Methods section, the cost in Greenland of lost IQ due to methyl mercury toxicity of children born is estimated to 59.1 million US$ each year (Table 1). The Greenlanders are bearing this cost without any gain from lower costs e. g. for reduction of Hg emissions as is the case in the USA, because nearly all Hg originates from outside their territory.

Table 1
Cost attributable to loss of intelligence from methyl mercury contaminated food in Greenland

Variable	Segment of births (percentile)						Sum
	27.2	33.5	47.4	74.5	98.2	>98.2	
Cord blood concentration (μg L^{-1})[a]	5.8	−11.6	−23.2	−46.4	−92.8	>92.8	
IQ-pints lost within interval	0	0.76	2.26	3.76	5.26	6.72	
Number of children affected[b]	–	61	134	262	229	17	703
Lost income (million US$)[c]	0	1.0	6.8	21.9	26.8	2.6	59.1

[a] Range 2.4–181 μg L^{-1}.
[b] 967 children born year^{-1} in average for 1998–1999 (UN, 2002).
[c] Loss of 1 IQ-point is valued to 22 275 US$ decreased lifetime earnings (see Material and methods section).

Some may argue that using a value of lifetime earnings calculated for the USA is inappropriate, because the value may be lower in Greenland. Certainly, the economic situation varies between countries but the per capita gross domestic product (GDP) demonstrates only a marginal difference between the USA (36 924 US$) and Denmark (39497 US$), administering Greenland (2003 US$; UN, 2004; no data are available on Greenland separately). In addition, USA is the dominating Hg emitter of the countries having territory in the Arctic and Hg emitted causes in health terms the same damage if a Greenlander is affected as if the victim is USA citizen.

The health effects most often associated with Hg toxicity are neurodevelopment deficiencies in developing fetuses and, at higher exposure, brain and other nervous damages in adults, but cardiovascular diseases may be even more costly (Rae and Graham, 2004; Rice and Hammitt, 2005). However, existing data from the Arctic has not permitted a more thorough study of health costs. For the same reason, also other costs related to Hg deposition in the Arctic have been omitted. Examples are lost recreational values and culture losses, physical stress associated with guidelines for consumption of fish and maritime products, and a possible reduction of biodiversity.

3.1.2. Aspects of mercury and related costs in the Arctic

Conventional economic approaches, such as cost benefit analyses, cannot handle fundamental differences between fishery and energy production via fossil fuel combustion. Fishery is an everlasting, renewable food resource as long as it is not harmed by pollution or excessive landings, while energy production via fossil fuel combustion is a non sustainable activity, extracting and finishing limited resources. This is why evasive efforts to not pay the full environmental costs should be counteracted so that the activities do not cause damages lasting far beyond the time limit of benefits obtained.

Smoke stacks at power plants are tall in order to distribute Hg and other pollutants emitted over a wider area and reduce deposition of them in the vicinity of the plant. A large part of Hg emitted to the atmosphere is transported globally as gaseous elemental mercury (Hg^0) before deposition, thereby shifting the pollution costs away from the ones profiting on no or reduced flue gas cleaning costs. Ethical aspects need to be considered when judging whether this is justified (O'Neill, 2004).

Small amounts of Hg enter the marine food web, are biomagnified and eventually end up in people. How little? Let us consider that there is about five liters of blood in the adult human body, which with data from the Hansen and Pedersen (1986) survey show that more than 80% of the population in north Greenland exceeded 250 μg Hg per person and 16% exceeded 1000 μg Hg per person. If one applies the lower value as an average value for 80% of the entire population of Greenland of approximately 50000 (Greenland statistical yearbook, 1992) and applies the 1000 μg limit to represent the average value of the remaining 20%, then the total amount of Hg reaching the top of the food chain in Greenland is 10 g in 80% of the population and 10 g in the other 20% of the population. This is totally 20 g of Hg distributed throughout the bodies of all Greenlanders, or $1/10^7$ of the approximate total annual Arctic deposition.

Increased atmospheric deposition of mercury since preindustrial times have increased lake and sediment Hg fluxes by a factor of 2.5–3 (Fitzgerald et al., 2005; Semkin et al., 2005). How much of the deposition that actually ends up in the biota is not fully understood. Most Hg deposition is as inorganic Hg but there is also a significant fraction of methyl Hg deposited, suggested to be from a marine source (St. Louis et al., 2005). In freshwater lakes in the Arctic, methylation is driven by atmospheric Hg deposition, but minor amounts of the methyl Hg formed enter the biota as the sink, while the major part is photo-decomposed (Hammerschmidt et al., in press). However, it is clear that other biological compartments, besides humans, are also acting as sinks, and above a certain concentration also paying a cost. This threshold concentration differs depending of type of damage and varies with species and individual susceptibility. For this and other obvious reasons, the evaluation is not easy, but the example illustrates the need to carry out careful, multi-discipline analysis, including non-monetary economic aspects, since only a very small fraction of the deposited Hg ends up in humans, and it is assumed that practically any amount of Hg in humans above normal background exposure is too much.

Mercury concentrations of Arctic, marine biota indicate an increasing trend (Muir et al., 1999; Riget et al., 2004), although the ability of time series available to detect trends is rather poor. Time series are needed with more frequent sampling and starting before the 1970's. One possibility could be to analyze Hg content of baleen kept at museums or found at archeological sites. Baleen is an incrementally-growing tissue of balaenopteran whales, which preserves relatively well over time and might be useful for studies examining long-term changes of metal levels in whales (Hobson et al., 2004). Also more comprehensive Hg time series analyses are needed for terrestrial biota and freshwater fauna to evaluate a possible relation to increasing Hg levels in sediment records of the Arctic (Braune et al., 1999).

Global warming will increase the Hg load in the Arctic due to melting ice and increased weathering as a result of smaller areas with permafrost, while the increased temperature will stimulate Hg methylation, and an increased content of organic matter in water will inhibit photodecomposition and extend its residing time in the water column. Thereby, natural sources, or reemission of originally anthropogenic Hg, may in the near future be a larger contributor to Hg becoming bioavailable in the Arctic than present, anthropogenic Hg emissions (Macdonald et al., 2005). However, it should be observed that the anthropogenic emissions can be controlled by human actions now, while Hg released due to global warming are in practice not controllable any longer, although the rising temperature is caused by anthropogenic emissions of greenhouse gases, mainly carbon dioxide (Paeth et al., 1999).

Atmospheric mercury concentrations have been nearly constant for the last half of the 1990's (Slemr et al., 1977). As the economy of China, globally the dominating Hg polluter, grows it will need to generate more power. Assuming it does this by increasing output of coal combustion power plants or building more plants with its present technology, then Hg deposition to the Arctic from this source will steadily increase in the future. Several new coal combustion power plants are also planned in the USA to compensate the decreasing energy output from oil, projected from the decrease in discoveries of new oil fields (Aleklett and Campbell, 2003).

Anthropogenic emissions of Hg, even if locally smaller than natural Hg sources, are adding to the natural Hg pool. Mercury background concentrations in biota are the result of equilibriums within different Hg cycles reached throughout millennia. Nowadays, large anthropogenic Hg emissions to air and water have resulted in Hg concentrations in biota increasing as an effect of efforts to reach a new equilibrium in chemical reactions and biological processes. The methylation cycle is poorly understood, especially in marine environments and quantitative links between the source and the effect are presently not available other than on a local or regional basis. However, it is evident that continued large Hg emissions from coal combustion will influence methyl Hg levels in Arctic biota and thus human exposure.

Stakeholders interested in continued exploitation of nature as a sink for contamination, by virtue of their ambition of maximized instant profit, are often disregarding externalities such as Hg bioaccumulation in biota and, as a consequence, advocating that their dissipative activities should not be hindered as long as it has not been scientifically proven that their emissions are the cause of a specific contamination driven damage. Considering the extraordinary powers of technology and that Hg is toxic and an element and therefore not degradable to harmless elements, an alternative approach, often referred to as the "precautionary principle", could be that Hg emissions would not be permitted until it eventually has been scientifically proven that they will not cause damages in any aspect.

3.2. Remediation of contaminated sites

The Swedish environmental protection agency has investigated the possibilities to remediate some extensively polluted industry sites in order to obtain the goal to hand over a clean environment to future generations. Mercury has been emitted at several of these sites. Here we present results from prospecting or remediation activities at the Swedish sites Delångersån, a river dewatering into the lakes Kyrksjön and Långsjön, the chlor–alkali plant at Skoghall (northern Vänern), the lakes Svartsjöarna and Turingen, and the bay Örserumsviken of the Baltic Sea. We also present experiences from Minamata, Japan. The presentations are focusing on economic data in relation to pollutants removed, since the economic aspects of remediation are usually not easily accessible, although they, generally, determine whether a remediation project will be executed or not.

3.2.1. Delångersån, Kyrksjön and Långsjön

Forså paper mill has been producing paper pulp and paper from 1868 until 1983 and emitted its effluents into the river Rolfstaån, a part of the river Delångersån, in Hudiksvall municipality, Gävleborgs county in central eastern Sweden, nearly 300 km to the north of Stockholm (Fig. 1; Braf and Johansson, 1996). Large numbers of cellulose fibers were emitted with the process water and these have built up large fiber banks, contaminated with Hg and chlor organic compounds. An organic Hg fungicide (phenyl mercury acetate) was used as an anti-slime agent in tubes and as a preservative for the pulp from the early 1940s until 1966. As a result, an estimated 2 tons Hg entered the river. The chlor organic compounds originate from recycled paper and transformer oils containing chlorine-based substances (PCB) and possibly also from wood impregnation chemicals such as pentachlor phenol. In a 1990 study, a 3-km stretch of the river contained 640 000 m^3 of fiber sediments, containing about 100 kg of Hg and about 10 kg of PCB (Braf and Johansson, 1996). The Hg content varied between 0.01 and 3.2 mg kg^{-1} dry matter. It was highest at the sediment surface and increased downstream. Methyl Hg made up 0.5–4.8% of the total Hg content. As a result of erosion and gas production, the fibers and pollutants are continuously being transported downstream and influencing ever-larger bottom areas in the river and the lakes Kyrksjön and Långsjön, situated downstream.

In 1966–1970, 1-kg pikes from Kyrksjön had on average 7.1 mg Hg kg^{-1} f.w. and the maximum values recorded were 14 and 20.4 mg Hg kg^{-1} f.w. (Braf and Johansson, 1996). Thereby the lakes have the dubious honor of world record for Hg in fresh water fish. The first years after that the Hg emissions were terminated, Hg content in fish decreased rapidly. In 1990, 1-kg pikes from Kyrksjön contained in average just below 2 mg Hg kg^{-1} f.w. and 0.2–0.3-kg perches from Kyrksjön and Långsjön contained in 1993 about 1 mg Hg kg^{-1} f.w. (Braf and Johansson, 1996). The decrease rate in fish Hg content has then approached an asymptotic shape and it was concluded that it would take centuries for the fish Hg content to reach background levels (0.5 mg Hg kg^{-1} f.w. in 1-kg pikes) without any restoration activities. Therefore, a restoration plan was made.

In Rolfstaån, it was estimated that 640 000 m^3 of contaminated fiber banks and bottom sediment with about 100 kg Hg and 10 kg PCB needed to be removed from the river and disposed safely on land to efficiently reduce the methylation and release of Hg from the sediment to the water column. The total cost for the remediation was estimated to 11–13 million US$ nearly a decade ago, corresponding to close to 150 000 US$ kg^{-1} Hg secured (Table 2). So far, the remediation has not been executed in Rolfstaån, because of the large costs and unpredictable outcome of remediation of just the river but not the lakes Kyrksjön and Långsjön (M. Palm, Länsstyrelsen Gävleborg, personal communication). Many years have passed since the pollution took place and contaminated fibers have been transported from the river and spread over large areas of the lake bottoms, which therefore now contain about 300 kg Hg. This means that even larger volumes of sediments must be removed for a successful remediation aiming at obtaining fish Hg levels approaching background values in a near future.

3.2.2. Skoghall

Skoghall chlor–alkali plant was built at the northern shore of the lake Vänern, just to the south of Karlstad, in 1918 and operated with Hg cells until 1989 (Fig. 1; Lundgren, 2001). An estimated 100–130 tons Hg has been emitted from the plant,

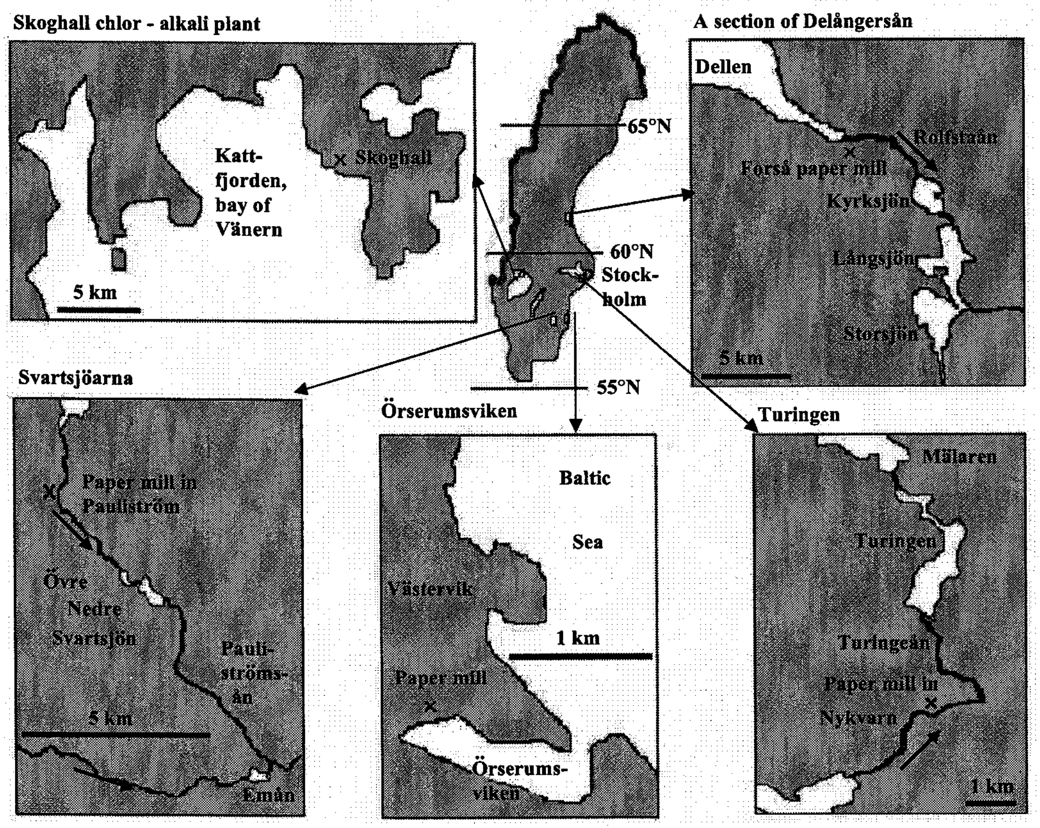


Fig. 1. Sites (rivers, lakes, and a bay of the Baltic Sea) polluted with mercury from a chlor–alkali plant at Skoghall and from paper mills at the other sites. Arrow beside a river indicates flow direction.

about half via water. This has resulted in that fishes, such as pikes, in adjacent parts of Vänern have Hg levels far above the limit for consumption. A successive change of chlor and alkali production for Hg free membrane technology was initiated in 1977 and completed in 1989, when Hg left over was transferred to another plant. The empty, contaminated building and soil is annually emitting about 16 kg Hg to air and 2.5 kg Hg to water (Lundgren, 2001).

Plans have been made to demolish the building and build a landfill for building scraps and stored wastes from the operation and contaminated soil, thereby reducing annual Hg emissions to an estimated one kg to the air and 0.5 kg to the water (Lundgren, 2001). However, the plans have still not been executed, because the authorities have not approved them. The lack of knowledge, laws, and instructions on how to handle this type of polluted industry site is a problem, delaying its remedy. The company wants to reduce the immediate emissions for a comparably low monetary cost, while the authorities, ideally, prefer remediation to original conditions and, as a minimum, want the emissions to be monitored also after the waste has been put in a landfill. The landfill is planned to contain 10 000 m^3 Hg contaminated material with an estimated total of 5400 kg Hg. No detailed budget has been made for the project, but the total costs have been estimated to 16 000 US$, corresponding to 3000 US$ kg^{-1}

Table 2
Costs to remediate mercury contaminated sites

Country	Site	Hg emitted (ton)	Hg to secure/ secured (kg)	Total cost (million SEK)	Total cost (million US$)	Cost (US$ kg^{-1} Hg secured)	Year of cost calculated	Observations
Japan	Minamata	190–225	95 000–115 000		517	4500–5400	1993	785 000 m^3 sediment dredged
Sweden	Rolfstaån	2	100 [a]	80–100	11–13	105 000–135 000	1995	640 000 m^3 fiber
	Skoghall	100–130	5400	120	16	3000	2002	10 000 m^3 building scrap and soil
	Svartsjöarna		15–150	100–120	15–16	98 000–1 100 000	2004	260 000 m^3 fiber
	Turingen		350	66	9	25 000	2004	225 000 m^3 sediment
	Örserumsviken		750	115	15	20 000	2002	1400 kg PCB, 170 000 tons sediment

[a] Another 300 kg in lake sediments of Kyrksjön and Långsjön.

Hg secured (Table 2). The low cost per unit Hg secured compared to other remediation projects is caused by a highly concentrated Hg containing waste, because only the most severely polluted soil from the industry site has been planned to be put in the landfill. If all soil at the site should be put in a landfill or cleaned from Hg by retorting or some other process, the cost will escalate. The cost would be much higher and totally unrealistic, if the goal is set to restore soils surrounding the site to background levels, being orders of magnitudes lower than the present level. The costs do not include remediation of the sediment contaminated in Vänern, although there is a plan for isolating the sediment of Anholmsviken, the bay of Vänern receiving the effluents from the chlor–alkali plant.

3.2.3. Svartsjöarna

Svartsjöarna, named after its black water, are two lakes in Hultsfred municipality, the province of Småland, southern Sweden (Fig. 1). They were contaminated with Hg from a paper mill located in Paulistöm. As in Rolfstaån, large numbers of fibers were emitted here, too, forming fiber banks of 260 000 m^3. The amounts of Hg left in the sediments is comparably small, 10–100 kg in the upper lake, Övre Svartsjön, and possibly half that amount in the lake downstream, Nedre Svartsjön (Olof Regnell, Lund University, personal communication). Thereby the remediation cost per unit Hg secured is high, up to 1.1 million US\$ kg^{-1} (Table 2). However, the environmental impact of Hg present is large because of chemical characteristics of the lake water, having a high concentration of dissolved organic matter. Such black waters are common in the boreal forest zone and known for increasing the fish Hg concentrations (Meili, 1991; Meili, 1997). The prospective for reduced fish Hg concentrations after remediation are good and the lakes are attractive for out-door activities. Starting in 2005, the lakes will be remediated for a planned cost of 15–16 million US\$, of which the former and present owners of the paper mill will contribute with about 20%, with the rest covered by government and municipal funds (Holmen et al., 2004).

3.2.4. Turingen

The lake Turingen is situated about 40 km to the southwest of Stockholm and drains into Mälaren, the main drinking water source of Stockholm (Fig. 1). A paper mill in the nearby municipality of Nykvarn used Hg fungicides between 1946 and 1966 (Projekt Turingen, 2003). An estimated 450 kg of Hg has contaminated Turingen and the river leading to the lake from the paper mill. The amount of Hg used in the plant is not known, but is expected to be many times larger, because most of the Hg used left the plant with wallpapers and other paper products sold.

The river and lake were remedied in 1995 and 1999–2003, respectively, at a cost of nearly 9 million US\$, corresponding to 25 000 US\$ kg^{-1} Hg (Table 2; Bergman, 2004). The works included digging a partly new canal for the river beside two ponds, where most of the 100 kg Hg found in the river system was accumulated. The eastern part of the inlet bay of the lake was dredged and contaminated sediment and reed roots were placed in the western part of the bay, covered by a synthetic fabric and a 0.4-m layer of clean, fine sand. Finally, 80% of the lake bottom was covered with an aluminum gel by applying $AlCl_3$ and NaOH with special equipment. The idea is that the gel of artificial aluminum polymers will reduce Hg water pollution by reducing the exchange between contaminated sediment and lake water and that the gel will reduce bioturbation and other sediment mixing processes between contaminated sediment and non-contaminated sediment formed on top of the gel. The formation of new sediment above the gel is crucial for long-term burial of the contaminated sediment, so the technology is not feasible in waters with a low sedimentation rate. The main advantage of the method is a lower cost than removing the sediment and placing it in a landfill. The long-term effect of this new technology is not known, but a 5-year assessment program commenced in 2004.

3.2.5. Örserumsviken

Örserumsviken, a bay of the Baltic Sea, just south of Västervik township, housed Westervik paper mill, which started its operation in 1915 and closed down in 1980 (Fig. 1; Jansson, 2003). Process water was let out into the bay, initially untreated, later, inadequately treated. As a result, the inner part of the bay filled up with fibers contaminated with Hg and PCB. Phenyl Hg acetate was used in the plant from the beginning of the 1950s until 1966. PCB originated from self-copying paper. In 1972, PCB was prohibited to use in open systems, but it continued to be emitted from the mill for many years, entering the production via recycled paper from archives (Meili, 2002).

Before closing down the plant in 1980, 220 000 m^3 of Hg contaminated fiber sediments from 15 ha of the 37 ha wide bay bottom was dredged and deposited on land. This as a step to remediate Örserumsviken, where swimming had been prohibited for health reasons and fishing prohibited because of high Hg content. However, Hg content in pike did not decrease after the remediation in 1978–1979. In 1986–1987, the fish were found to also contain PCB. A third environmental pollutant, poly aromatic hydrocarbons (PAH), was discovered in 1997 (Meili, 2002).

A second remediation was therefore carried out in Örserumsviken in 2001–2003, because the first one had been incomplete and not reduced the pollution to desired levels. In addition, the problem with PCB and PAH had not been considered the first time. The second remediation has now been completed, including dredging the major area of the bay and securing the open-air landfill from the earlier remediation. The total costs amount to 15 million US\$, corresponding to 20 000 US\$ kg^{-1} Hg secured (Table 2). Assigning the whole remediation cost on the quantity of Hg secured may seem illogical, because avoiding environmental effects of PCB was just as important a reason for the remediation, as securing Hg. Therefore, possibly only half of the costs should be assigned to secure Hg. This results in a cost of 10 000 US\$ kg^{-1} Hg secured. However, the remediation costs in 1978–1979 were not included. They were 8.3 million SEK (1.1 million US\$) in 1979, today corresponding to about 4 million US\$ index-corrected value (SCB, 2004).

3.2.6. Minamata

Minamata, a fishing village in the south of Japan on the Minamata bay, is the site of one of humanity's most tragic cases of industrial pollution. In the 1940s, it was observed that rotten oysters became increasingly apparent and dead fish were found floating in the bay. In 1951, seabirds were dropping from the air. In 1953, the village cats could not walk steadily and the unknown disease afflicting the village was called the dancing cat disease. One year later, the deadly "dance" was recognizable in humans, who were mainly nourished by marine products from the bay and the name was changed to Minamata disease, which also turned out to be the diagnosis of a man getting ill already in 1942 (Environment Agency of Japan, 1994; Mari Susa, Kumamoto University, personal communication of a journalist's chronology). Already at an early stage, the cause was thought to be discharge from Chisso, a company established in Minamata a few decades earlier. In 1959, it was scientifically proven that organic Hg caused Minamata disease and that the Hg originated from the effluents of Chisso. Chisso used Hg salts ($HgSO_4$ and $HgCl_2$) in two production processes: acetaldehyde (an intermediate in plastics production) and vinyl chloride (another plastic). The wastewater contained both inorganic Hg as well as methyl Hg, originating mainly from the acetaldehyde process.

Despite the evident causes of Minamata disease, Chisso, supported by the Japanese government, continued to emit Hg into the sea until 1968, when wastewater from the vinyl chloride production was directed to a special pond for the three years prior to the production being halted in 1971 (Kudo and Turner, 1999). The acetaldehyde factory was closed in 1968, because the method had become outdated (Environment Agency of Japan, 1994). However, acetaldehyde is still produced using the same technology and with similar toxic emissions in China, showing that shortsighted economy still outweighs human and environmental health (Watts, 2001).

More than one hundred persons died as a direct result of methyl Hg poisoning in Minamata and tens of thousands were diagnosed with brain damage and damage to other parts of the nervous system, characteristic of methyl Hg poisoning. Therefore, mercury poisoning contributed to the death of many more persons. The symptoms ranged from deafness, blindness, paralyses and convulsions to unsteady walking, restricted field of vision and numbness (Tsubaki and Takahashi, 1986; Takeuchi and Eto, 1999). The economic compensation to victims poisoned with methyl Hg in Minamata has amounted to nearly 1500 million US$ in current values from the 1950s until October 2004, when the Japanese Supreme Court settled the last Minamata disease lawsuit and found, in agreement with the District Court and High Court previously, that Chisso and the central and Kumamoto prefectural governments shared responsibility for failing to control the disease (Environment Agency of Japan, 1994; Pollack, 1997; Murayama, 2004). The governments were ordered to pay a total of 71.5 million yen (652 900 US$) in compensation to 37 plaintiffs and Chisso was ordered to pay 240 million yen (2.2 million US$) $) in compensation in this lawsuit (Murayama, 2004). Once the responsibility of the governments had been settled in a lawsuit, additional economic compensation became available to victims (Arita, 2005).

From 1932 until 1968, an estimated 190–225 tons Hg were emitted with the wastewater into the ocean from Chisso plants in Minamata (Kudo and Turner, 1999). Compensation to Minamata disease victims has therefore amounted to 6300–7500 US$ kg^{-1} Hg emitted. Good health is invaluable and not possible to buy, once one has been poisoned by methyl Hg. However, only where there are large-scale monetary costs, as opposed to human costs, has the industry taken notice. This was experienced in Minamata and the same attitude is demonstrated nowadays, when industries with Hg stockpiles, notably the American and European chlor–alkali industry, dispose their stock to usages, which harm the health of the final buyers; such as gold miners using the amalgamation method. When considering the costs for depositing excess Hg in an environmentally sound Hg repository, it is as comparison, valuable to have some figures on health costs caused by Hg handled improperly. The calculations presented are not an attempt to quantify the total health costs due to Hg pollution in Minamata, but only to present some figures obtained from available data. The total health costs are not possible to estimate until the full extent of the effects caused by Hg emissions from Chisso have been revealed. The health effects were not restricted to Minamata Bay and around the Minamata River, but were extended to the major part of the population of about 200 000 persons living around the inland Sea of Shiranui at that time (Fig. 2; Ninomiya et al., 2005). In the co-ordinate efforts of Chisso and the Japanese government to hide the extent of methyl Hg poisoning, only 2265 persons were authorized as Minamata disease patients and eligible for full compensation, while another 12 000 persons received limited compensation (Environment Agency of Japan, 1994; Watts, 2001). Also the latter were being damaged by methyl Hg from the factory but they were lacking one or more criteria prescribed for Minamata disease, criteria set up by economic and political reasons rather than out of objective, clinical criteria. Instead of launching a survey of the real extent of Minamata disease, the

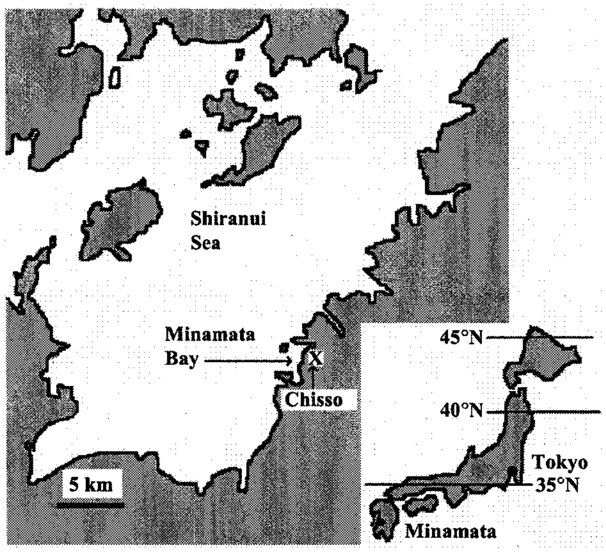


Fig. 2. Waters polluted with mercury from Chisso chemical plant in Minamata, Japan.

Japanese government is trying to conciliate certain victims with economic benefits (Arita, 2005). An approach which may hinder the determination of the full extent of effects at Minamata.

Minamata disease was the direct cause of 101 deaths and a contributing factor to another 800 deaths (Tsubaki and Takahashi, 1986; Watts, 2001). A human life is valued at 2.19 million US$ (16.3 million SEK) when studying the willingness to pay for safer roads in Sweden (SR, 2005). Using this value for the deaths in Minamata results in totally 221 million US$ for the 101 deaths and another 1752 million US$ for the other 800 deaths. This corresponds to about 10 000 US$ kg^{-1} Hg emitted (980–1160 US$ kg^{-1} Hg for the 101 deaths and 7780–9220 US $ kg^{-1} Hg for the other 800 deaths), which is more than the compensation paid to Minamata disease victims.

Commercial fishing was also severely affected by the effluents from Chisso, but the compensation paid to fishery has been relatively moderate, at about 90 million US$, corresponding to about 400 US$ kg^{-1} Hg emitted (Environment Agency of Japan, 1994), while the Minamata Disease Municipal Museum (2000) presents a somewhat lower figure (about 80 million US$). The comparatively low value is an effect of the authorities' late ban on fishing and no ban on fishing in all waters where the fish were found to have unsanitary Hg levels (Environment Agency of Japan, 1994; Ninomiya et al., 2005). As a result, many more persons were poisoned than would have had been the case with a more accurate response from the authorities on the high Hg levels in fish. In short, by reducing monetary losses for commercial fishing, health costs in the region were drastically increased.

In 1971, the planning for remediation of Minamata bay started (Kudo and Turner, 1999). A plan was set in 1975 and work commenced in 1977, but was suspended until 1980, when it was continued and finally completed in 1990. A steel and concrete wall was built, dividing the bay in two parts. All sediment contaminated with Hg above 8.75 mg kg^{-1} (and part of sediment with 5.0 to 8.75 mg kg^{-1}) outside the wall was dredged and pumped to the inside part of the bay for burial below fabric and a layer of virgin soil (Hosokawa, 1993; Kudo and Turner, 1999). An estimated 10–30% of emitted Hg had been redistributed into the sea outside the bay before dredging was initiated in 1980 (Kudo and Turner, 1999). At dredging, sediment polluted with Hg levels below 5–8.75 mg kg^{-1} was allowed to remain in the basin (Hosokawa, 1993). Therefore, we have estimated that only 50% of Hg emitted was secured by dredging and stabilization in the inner part of the bay. With this assumption, the remediation cost ranged between 4500 and 5400 US$ kg^{-1} Hg secured in Minamata Bay (Table 2). Of this sum, about 500 US$ kg^{-1} Hg are assigned to the Marushima/Hyakken Waterways and Marushima Port projects (Minamata Disease Municipal Museum, 2000).

3.3. Measures to avoid mercury pollution

In connection with remediation costs, it is valuable to study the costs for measures to avoid Hg emissions and related pollution. The actions may for practical reasons be divided between measures against pollution from intentional use of Hg, such as Hg pollution as a result of Hg used in dentistry and industry, and pollution from activities where Hg is not intentionally used, such as combustion of fossil fuels, where Hg is emitted because of its presence as a microelement in coal, oil, and gas.

3.3.1. Pollution from intentional use of Hg

Pollution caused by continued intentional use of Hg is easy to stop, because interrupted usage will result in terminated emissions and terminated pollution. The only major, intentional use of Hg presently lacking a technically and economically viable, Hg free alternative is the use of Hg in fluorescent tubes and other low energy lightning sources (Hylander and Meili, 2005). However, Hg free low energy lamps have been developed and are expected to enter the market in the near future.

Historical, intentional use of Hg has resulted in large stockpiles of Hg in society, which will result in continued pollution even after a total stop of using Hg in processes or in new products. Mercury used as electrodes in chlor–alkali plants is globally the largest stock of Hg and has been the second largest polluter of Hg used intentionally. The Hg cells in Sweden (400 000 kg) are due to be replaced by Hg free technology before 2010 and accompanied by a safe disposal of their Hg stock (Hylander and Meili, 2005). Mercury cells are well suited to be phased out at a faster pace globally, considering the comparatively low conversion costs per kilogram Hg (Table 3).

Mercury in dental fillings of the population is the second largest stock of Hg (about 40 000 kg) in Sweden. Besides these stocks, large amounts of Hg are in use in measuring and electrical equipments and in laboratory chemicals in most industrialized countries. Ongoing and potential pollution from these stocks are large in quantitative terms and manifested as many small emission sources over large geographic areas. This is perhaps most evident when looking at dental amalgam, which is carried by 74% of the grown up population in Sweden and results in a continuous release of about 100 kg Hg per year to the wastewater via every day chewing (Skare and Engqvist, 1994; KemI, 2004). This demonstrates that it is not possible to stop pollution from this Hg stock. In addition, technologies to reduce the emissions are costly and more difficult than replacing Hg as a dental filling material.

Replacement of Hg containing products in use for Hg free ones is a possible strategy to prevent pollution from other parts of the Hg stock in society. This strategy has been successfully employed, as demonstrated by the more than 345 000 potential pollution sources in the form of Hg thermometers having been replaced by Hg free thermometers in Sweden (Rein and Hylander, 2000). Where the historical usage and localization of Hg containing products is poorly known, employment of elder persons, earlier engaged in installing Hg containing equipment in e.g. real estates, is a possible course of action for tracking and document the localization of Hg containing products. Results from Sweden and estimates from Minnesota indicate that the costs to remove potential pollution sources, such as Hg containing equipments and laboratory chemicals,

Table 3
Costs for strategies avoiding Hg pollution and their potential to reduce Hg pollution, expressed in the classes: small, medium, and large

Activity	Place and year	Cost[a] (US$ kg^{-1} Hg)	Reduction potential	Reference
Return of Hg thermometers	Sweden, 1992–1996	950–1200[b]	Large	Rein and Hylander, 2000
Replace mercury-containing items	Minnesota, estimated 1999	20–2000[c]	Large	Jackson et al., 2000
Collect Hg and Hg compounds in school labs	Sweden, 1995–1999	70–400[b]	Small	Rein and Hylander, 2000
Collect metallic Hg in school laboratories	Minnesota, estimated 1999	20[c]	Large	Jackson et al., 2000
Collect Hg compounds in school laboratories	Minnesota, estimated 1999	1400[c]	Small	Jackson et al., 2000
Replacing Hg cells at chlor–alkali plants	USEPA, estimated 1996	10 100[d]	Large	USEPA, 1997
Increase recycling of chairside traps in dentistry	Minnesota, estimated 1999	240	Medium	Jackson et al., 2000
Install amalgam separators	Minnesota, estimated 1999	33 000–1 300 000	Medium/Large	Jackson et al., 2000
Replace dental amalgam fillings at dentists	Sweden, estimated 2004	129 000	Large	This study
Remove dental amalgam fillings at death	Sweden, estimated 2004	400	Large	This study
Flue gas cleaning with carbon at crematoria	Sweden, estimated 2004	170 000–340 000	Medium/Large	This study
Flue gas cleaning with carbon at crematoria	UK, estimated 2004	29 000	Medium/Large	This study, BBC News, 2005
Medical waste incinerators with scrubber	USEPA, estimated 1996	4400–8800	Medium/Large	USEPA, 1997
Carbon injection into flue gases at waste incinerators	USEPA, estimated 1996	465–1900	Medium/Large	USEPA, 1997
Combined technologies at waste incineration	Uppsala, Sweden, 2004	40 000	Large	This study
Coal cleaning, conventional, chemical or both	Minnesota, estimated 1999	100 000–128 000	Large	Jackson et al., 2000
Carbon injection into flue gases at power plants	USEPA, estimated 1996	31 000–49 000[e]	Large	USEPA, 1997
–"–	US Dep. Energy, estimated 1996	149 000–154 000[e]	Large	Brown et al., 2000
–"–	Minnesota, estimated 1999	20 000–725 000	Large	Jackson et al., 2000
Combined technologies at power plants	USEPA, estimated 1996	11 000–61 000[e]	Large	USEPA, 1997
–"–	US Dep. Energy, estimated 1996	56 000–85 000[e]	Large	Brown et al., 2000
Wind as replacement for energy from coal	Minnesota, estimated 1999	1 200 000–2 000 000	Large	Jackson et al., 2000

[a] Values in a range reflect differences across facilities of different sizes or at different recovery rates e.g. 90% or >95% of Hg recovered from flue gases, or other site-specific conditions.

[b] Cost calculated per kilogram Hg collected and includes costs for information, reimbursement for thermometers, and additional costs for collecting, transport and deposition, while costs for additional working time of shop assistants, municipal officials, etc. are excluded.

[c] Total cost per unit of Hg not emitted.

[d] Capital and electrical costs. Indirectly reduced Hg emissions caused by lower consumption of electricity from Hg emitting power plants have not been included. The costs increase if pollution occurred earlier needs extensive remediation.

[e] 90% reduction in mercury emissions. The EPA figures are based on a lower flue gas temperature when carbon is injected, thereby using the sorption capacity better, resulting in that only 2–34% active carbon is used compared to the DOE estimates.

range between 20 and 2000 US$ kg^{-1} Hg (Table 3), which is from four times the registered Hg commodity price at the actual time to 400 times higher. It should be noted that the Swedish figures are the real costs to remove Hg from products in use, while the figures from Minnesota are estimated costs to reduce estimated emissions of Hg via the actual strategy.

A low cost per kilogram Hg removed/not emitted does not necessarily indicate that this strategy should get the highest priority if the economic resources for avoiding Hg emissions are limited. Other factors should also be considered. Such factors are potential damage of Hg emitted and, for the Swedish figures, potential emission from the actual Hg stock. These considerations have been summarized as the reduction potential of the actual strategy, defined as the amount of Hg release that can be prevented or collected (Table 3). The reduction potential of the studied replacement strategies is often large. An exception, when looking at immediate effects, is collecting Hg and Hg compounds, especially in Swedish school and university laboratories. This is because Hg was generally not used in the laboratory exercises at time of collection, but had been left from earlier periods, when the health and environmental effects of Hg were less recognized. In case the Swedish waste handling instruction would be followed at any future disposal of these compounds, the Hg would be safely handled and disposed as harmful waste in the future, while actual emissions from the compounds on the laboratory shelves were small. Nevertheless, collecting Hg in schools and universities got high priority in Sweden for reasons below.

Firstly, the pupils and students got involved in the collection campaigns, thereby resulting in increased awareness among the generation growing up, about mercury and environmental pollutants in general. This awareness was also transmitted to their parents and to the society in general, partly because large mass media attention about the employment of Hg tracker dogs. These dogs found Hg also where nobody knew there was any mercury, such as spills and Hg stored in irrelevant places without correct documentation. The risk that Hg may not be properly handled in the future, if not collected in a special campaign, was another reason that collecting Hg was organized in national school and university campaigns. The risk of improper handling of Hg waste is mainly not caused by nonchalance or illegality, but it is rather due to human limitation to be correctly informed about proper handling of thousands of chemicals in our daily lives, where for example, just one Hg compound, phenyl mercury acetate ($C_8H_8HgO_2$), is traded under at least 75 different names (The Registry of Toxic Effects of Chemical Substances as referred in Maxson, 2004).

Mercury emissions from earlier dental amalgam fillings will continue for several decades after a change to Hg free filling materials. Installing amalgam separators at dental clinics and advanced flue gas cleaning at crematories will reduce the major part of the emissions from these sources. The related investment and running costs for these installations should be included in the cost for using dental amalgam. Presently, this is not the case in any country worldwide. As a consequence, amalgam fillings are considered to be economic while they de facto are more expensive than most, possibly all, other fillings when including environmental costs (Table 3).

Mercury emissions to wastewater from dental clinics can be reduced by installing amalgam separators at different cost levels (Table 2). The less expensive ones are working according to the sedimentary principle and can recover about 85% of Hg emitted to wastewater from dental chairs, while an additional device, recovering suspended and dissolved Hg may result in that more than 99% of the Hg emitted is recovered (Hylander et al., in press). A technical break-through is underway, so the high investment costs for amalgam separators resulting in high costs per kilogram Hg recovered are expected to decrease markedly in the near future and is presently markedly lower in Sweden than the estimates from Minnesota as presented in Table 3.

It should be observed that emissions of Hg to wastewater from everyday chewing cannot be avoided in any other way than replacing amalgam fillings with Hg free fillings. Although technically possible, this is expensive at 129 000 US\$ kg^{-1} Hg, calculated as the average cost to replace an amalgam filling containing 0.6 g Hg with a composite filling at Uppsala county council's healthcare service (Folktandvården i Uppsala Län, 2004). We have in this cost not considered any possible health benefits associated with removing the fillings of persons with certain autoimmune and allergic diseases (Shimazu-Kohdera, 2000; Prochazkova et al., 2004).

Amalgam fillings not replaced before death will cause emissions to air, soil, and water upon cremation or burial. Mercury emissions from crematoria are one of the largest air pollution sources in many countries and several crematoria are being equipped with flue gas cleaning technology with associated, high costs (Table 3). The technology cannot recover all Hg in the flue gases, and a certain pollution level of Hg will still occur, contrary to if the fillings are removed before cremation. Removing amalgam fillings in an aesthetically correct and environmentally friendly way is less intruding or offensive than for example, the routine post-mortem examination and removal of a pacemaker in a person who utilized one. No investment is needed in infrastructure and the costs are restricted to working time spent. In addition to being an economically advantageous way to eliminate all Hg emissions from dental amalgam at both cremation and burial, the technology is easy to learn and can be used in all countries, regardless of economic and technical conditions.

3.3.2. Pollution of Hg from combustion

Pollution of Hg from combustion of fossil fuels is the dominating source of Hg pollution. Coal combustion is currently the main source of these emissions, worldwide contributing between 750 (Pirrone et al., 1996) and 1500 tons per year (Pacyna and Pacyna, 2002), thereby responsible for 3/4 of all current anthropogenic Hg emissions to the global atmosphere. Also oil and nature gas contain Hg emitted at combustion if not removed beforehand as generally done for gas, in the Netherlands yielding 5–10 tons Hg annually (UNEP, 2002). The mercury pollution from combustion of fossil fuels has problems in common with incineration of wastes (also called waste combustion) and cremation, where the Hg emissions are mainly the result of intentional use of Hg. These problems are a distant distribution of certain species of Hg emitted and that different species (particle bound, Hg^{II+}, and Hg^0) of Hg are emitted. Especially gaseous, elemental Hg (Hg^0) is transported far after emission, in average three turns around the globe before deposition (UNEP, 2002). Since a large portion of Hg emitted from combustion creates problems outside local and national boundaries, the interest is limited from the polluters to combat Hg emissions from combustion. At least by other means than using electrostatic precipitators to collect ash particles with particle bound Hg, which otherwise will deposit locally and result in blackening as the ash particles deposit.

The costs for flue gas cleaning is comparably high compared to other pollution reducing strategies (Table 3). One reason is the relatively low concentration of Hg combined with large amounts of material handled. Another is that a combination of different techniques is needed to efficiently capture the different Hg species in flue gases. Installing cleaning systems in an old plant is more expensive than if it is included initially when built, especially if a scrubber is to be installed, and may give sub-optimal cleaning efficiency for some pollutants other than Hg. Therefore, all new generation sources using fossil fuels and waste incinerators should be equipped with efficient flue gas cleaning systems at the time of construction. Although this will increase the investment capital cost by about one third, this is the most cost effective way to reduce global pollution of not only Hg, but of other pollutants from these sources. In addition to the control of mercury emissions there are other benefits presented below.

A new waste incinerator, with capacity to incinerate 175 000 tons solid waste per year and using the heat produced in the district heating network and supplying steam to nearby industries, has been demonstrated in Sweden, It was completed in Uppsala in 2004 for about 135 million US\$, of which 40 million US\$ was spent on flue gas cleaning equipment (Sollenberg, 2004, personal communication). Assuming 20 years of operation, 0.3 g Hg ton^{-1} waste and 96% removing efficiency (Table 4), 1000 kg of Hg will be recovered with an investment cost of 40 000 US\$ kg^{-1} Hg. In addition, several other metals, arsenic, sulfur, and chlorine will also be recovered and there will be practically no emissions of dioxins (Table 4, Hylander et al., 2003). In order to simplify the calculations, these pollutants are not bearing the investment cost in this example, but have been assumed to bear maintenance and other running costs. The running costs include chemicals used for cleaning wastewater from the scrubber and landfill costs for used active carbon.

Combining three devices (electrostatic precipitator or ESP, scrubber/condenser, and Filsorption unit with carbon injection) results in higher investment costs than using a single or two devices, but is the only approach available to capture different Hg species (USEPA, 1997; Hylander et al., 2003). In addition to a higher recovery rate for several pollutants, the amount of

Table 4
Recovered and emitted metals and arsenic from waste incineration at Uppsala 2001–2003[a]

Element	Content (g ton^{-1} waste wet weight)	Recovered[b] in bottom ash (%)	Emitted[b] from flue gases (%)	To air (%)	To water (%)
Mercury	0.27	1.32	96.2	2.41	0.078
Lead	341	83.05	16.94	0.004	0.002
Cadmium	506	29.44	70.53	0.013	0.017
Chrome	23	83.44	16.59	0.019	0.002
Nickel	18	92.64	7.32	0.018	0.014
Zinc	636	55.54	44.45	0.006	0.004
Cobalt	3.8	88.31	11.66	0.004	0.030
Arsenic	2.1	50.83	49.09	0.079	0.005
Copper	432	96.08	3.84	0.001	0.001

[a] For description on sampling and analytical procedures, see Hylander, Sollenberg, and Westas, 2003.

[b] The sum may slightly deviate from 100% because only two-three decimals are presented.

carbon used in the cleaning is drastically reduced when combining these devices. The operating as well as landfill costs are thereby reduced (Table 3) and positively accompanied with reduced risk of future Hg losses from landfills. The advantage of having a less voluminous Hg containing waste is also an important advantage when considering the case of tooth extraction rather than flue gas cleaning at crematoria.

The Hg content of household waste incinerated at Uppsala is low in an international context (Hylander et al., 2003) and incinerating waste with inadequate separation of Hg containing items, such as batteries, energy saving lamps, and fluorescent tubes, will result in a higher content of Hg in the waste and subsequently lower cleaning cost per unit Hg removed, typically 5000–10 000 US\$ kg^{-1} Hg (Poulson, 1994) or 500–2000 US\$ kg^{-1} Hg, if a lower recovery rate and larger emissions are accepted than in the strict emission control at Uppsala (Table 3; USEPA, 1997). These figures may seem more appealing than the costs to remove Hg from power plants in operation (Table 3). The reason for the higher costs for removing Hg from power plants compared to waste incineration is partly a result of comparatively low Hg concentrations in coal. Another is the lower content of hydrogen chloride (HCl), resulting in the formation of mercuric chloride ($HgCl_2$), which is easily removed from flue gases of waste incinerators. Also, the high content of sulfur in coal and the subsequent formation of sulfur dioxide (SO_2) in the flue gas hampers Hg removal because it can act as a reducing agent, converting oxidized mercury to elemental mercury. These problems may be reduced by co-firing coal with other fuels containing more chloride and less sulfur.

Other pollutants, removed simultaneously as Hg with the same equipment, have not been considered in the presented power plant examples. These pollutants, ranging from other toxic elements and compounds to acidifying agents such as oxides of sulfur and nitrogen, need to be removed to safeguard our environment and human health. Large amounts of mercury, cadmium, and arsenic are leaving the flue gases if no cleaning is performed, while copper, nickel, cobalt, chrome, and lead are mainly left in the bottom ash (Table 4). The cleaning costs and safe disposal of the ashes should be considered when projecting new, as well as when operating existing power plants and waste incinerators. The costs should be added to the energy cost or waste handling charge to avoid that we as consumers are adversely affecting the living conditions of future generations.

4. Conclusions

Efforts to reduce human and environmental exposure to Hg must be prioritized because of the adverse health and environmental effects. In a world of increasing population, such a nutritive food resource as fish and other seafood should not be withdrawn from human consumption due to anthropogenic pollution. Avoiding pollution is far more cost effective than remediation of polluted sites. Prevention combined with control measures is the only option to avoid further deterioration and a needed recovery of large and especially vulnerable regions such as the Arctic. Remediation costs in the presented case studies range between 2500 and 1.1 million US\$ kg^{-1} Hg isolated from the biosphere depending on local circumstances such as quantities to secure, nature of pollution, media, geography, technology chosen etc. In general, remediation costs are lower the sooner remediation takes place after the pollution has occurred. To prevent pollution, regulations on discontinued use of Hg is cost effective and should be combined with extensive flue gas cleaning for all power plants and waste incinerators on a global basis.

Acknowledgements

Work supported by Foundation for Metal Biology, Uppsala. We would like to thank all persons contributing to this article, especially Hans Sollenberg and Håkan Westas, Vattenfall AB, Uppsala, Tom Lundgren and Rune Wettström for data on Skoghall, Martin Palm, Länsstyrelsen Gävleborg, Olof Regnell, Lund University, Ronald Bergman, Project Turingen, and Mari Susa, Kumamoto University, for data on Rolfstaån, Svartsjöarna, Turingen, and Minamata, respectively. Michael E. Goodsite thanks Mark H. Hermanson and William Shotyk for their support in visiting their respective institutes.

References

ACAP. Assessment of mercury releases from the Russian Federation. Copenhagen, Denmark: Russian Federal Service for Environmental, Technological and Atomic Supervision and Danish Environment Protection Agency for Arctic Council; 2005.

Aleklett K, Campbell C. The peak and decline of World oil and gas production. Miner and Energy 2003;18:5–20.

Arita, E. More Minamata victims to receive financial aid. Tokyo, Kumamoto say assistance program will be open to 3000 additional sufferers. The Japan Times: April 8, 2005. http://www.japantimes.co.jp/cgi-bin/getarticle.pl5?nn20050408a2.htm, accesssed 10 April, 2005.

BBC News. Crematoria warned over mercury. Strict rules for crematoria to limit mercury pollution caused when tooth fillings are vaporised have been announced by ministers. http://news.bbc.co.uk/go/pr/fr/-/2/hi/health/4160895.stm, published 1 Jan. 2005, accessed 10 Jan. 2005.

Bergman, R. Final Financial Report. Rapport till EU Kommissionen från Nykvarns kommun, 2004. http://www.turingen.com/bilder/final_financial_report.pdf updated 2004-11-01, accessed 15 October 2004.

Bjerregaard P, Hansen JC. Organochlorines and heavy metals in pregnant women from the Disko Bay area in Greenland. Sci Total Environ 2000;245(1–3):195–202.

Braf L, Johansson J-Å. Kvicksilver och organiska miljögifter i Rolfstaån-Delångersån (Mercury and organic pollutants in Rolfstaån-Delångersån.) Rapport, vol. 1. Gävle, Sweden: Länsstyrelsen i Gävleborg; 1996 [in Swedish].

Braune B, Muir D, DeMarch B, Gamberg M, Poole K, Currie R, et al. Spatial and temporal trends of contaminants in Canadian Arctic freshwater and terrestrial ecosystems: a review. Sci Total Environ 1999;230(1–3):145–207.

Brown TD, Smith DN, O'Dowd WJ, Hargis Jr RA. Control of mercury emissions from coal-fired power plants: a preliminary cost assessment and the next steps for accurately assessing control costs. Fuel Process Technol 2000;65:311–41.

Dastoor A, Larocque Y. Global circulation of atmospheric mercury: a modelling study. Atmos Environ 2004;38:147–61.

Egeland GM, Ponce R, Knecht R, Bloom NS, Fair J, Middaugh JP. Trace metals in ancient hair from the Karluk Archaeological Site, Kodiak, Alaska. Int J Circumpolar Health 1999;58(1):52–6.

Environment Agency of Japan. Our Intensive Efforts to Overcome the Tragic History of Minamata Disease. Environmental Health Department, Environment Agency of Japan; 1994.

FAO. 2004. FAO Fishstat Plus. http://www.fao.org/fi/statist/fisoft/fishplus.asp, accessed 10 October 2004.

Fitzgerald WF, Engstrom DR, Lamborg CH, Tseng CM, Balcom P, Hammerschmidt CR. Modern and historic atmospheric mercury fluxes in northern Alaska: global sources and Arctic depletion. Environ Sci Technol 2005;39:557–68.

Folktandvården i Uppsala Län. 2004. Prislista A 2004. Allmäntandvården (Pricelist for dental care). http://195.43.38.249/ftv/download/priser.pdf, accessed 10 October 2004.

Galváo LAC, Corey G. Serie vigilancia 7. Metepec, Mexico: Centro Panamericano de Ecologia Humana y Salud, Organización Panamericana de la Salud, Organización Mundial de la Salud; 1987 [in Spanish].

Grandjean P, Weihe P, Jørgensen PJ, Clarkson T, Cernichiari E, Viderø T. Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. Arch Environ Health 1992;47:185–95.

Grandjean P, Weihe P, Needham LL, Burse VW, Patterson Jr DG, Sampson EJ, et al. Effect of a seafood diet on mercury, selenium, arsenic, and PCBs and other organochlorines in human milk. Environ Res 1995;71:29–38.

Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, et al. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury — neuroradiologic and electrophysiologic studies. Neurotoxicol Teratol 1997;19(6):417–28.

Grandjean P, Budtz-Jørgensen E, Steuerwald U, Heinzow B, Needham LL, Jørgensen PJ, et al. Attenuated growth of breast-fed children exposed to increased concentrations of methylmercury and polychlorinated biphenyls. FASEB J 2003;17(6):699–701.

Greenland statistical yearbook. Nuuk, Statistics Greenland. ISSN 0907-9432; 1992.

Hammerschmidt CR, Fitzgerald WF, Lamborg CH, Balcom PH, Tseng CM. Biogeochemical cycling of methylmercury in lakes and tundra watersheds of Arctic Alaska. Environ Sci Tecnol [in press].

Hansen JC, Pedersen HS. Environmental exposure to heavy metals in North Greenland. Arctic Med Res 1986;41:21–34.

Hansen JC, Wulf HC, Kromann N, Albøge K. Human exposure to heavy metals in East Greenland: I. Mercury. Sci Total Environ 1983;26:233–43.

Hayes, M. Exotic mercury soars to high on tight supply. Reuters. 2004. http://www.reuters.com/newsArticle.jhtml?type=reutersEdge&storyID=6115119 updated 2004-08-31, accessed 2 September 2004.

Hobson KA, Riget FF, Outridge PM, Dietz R, Born E. Baleen as a biomonitor of mercury content and dietary history of North Atlantic Minke Whales (Balaenopetra acutorostrata): combining elemental and stable isotope approaches. Sci Total Environ 2004;331(1–3):69–82.

Holmen AB, Metsä Tissue AB, Länsstyrelsen i Jönköpings län. Holmen och Metsä Tissue bidrar till saneringen av Svartsjöarna (Pressrelease on remediation of Svartsjöarna). http://www.holmen.com/svenska_folder/news_center/index_nw1.html, released 2004-04-07, accessed 15 October 2004 [in Swedish].

Hosokawa Y. Remediation work for mercury contaminated bay— experiences of Minamata bay project, Japan. Water Sci Technol 1993;28:339–48.

Hylander LD, Meili M. The rise and fall of mercury: converting a resource to refuse after 500 years of mining and pollution. Crit Rev Environ Sci Technol 2005;34:1-36.

Hylander LD, Sollenberg H, Westas H. A three-stage system to remove mercury and dioxins in flue gases. Sci Total Environ 2003;304(1–3):137–44.

Hylander, LD, Lindvall, A, Uhrberg, R, Gahnberg, L, Lindh, U, Mercury recovery in situ of four different dental amalgam separators. Sci Total Environ. In press.

Jackson AM, Swain EB, Andrews CA, Rae D. Minnesota's mercury contamination reduction initiative. Fuel Process Technol 2000;65:79–99.

Jansson, T. 2003. The Bay of Örserum Project. Ready for remediation. The Executive Office of the Town Council of Västervik, Sweden, 2003. http://www.vastervik.com/miljo/orserum/rapport/Webbeng.pdf, updated 2003-02-17, accessed 15 April 2004.

Jernelöv A. Swedish environmental debt. Swedish government official reports series, vol. 126. Stockholm, Sweden: SOU; 1992. 39 pp.

Johansen P, Muir D, Asmund G, Riget F. Human exposure to contaminants in the traditional Greenland diet. Sci Total Environ 2004;331(1–3):189–206.

KemI. Report 4/04. Mercury — investigation of a general ban. KemI, October 2004. Report by the Swedish Chemicals Inspectorate in response to a commission from the Swedish Government. http://www.kemi.se/upload/Trycksaker/Pdf/Rapporter/Rapport4_04.pdf, accessed 10 October 2004.

Kava R, Noelle Stimola A, Weiser R, Mills L. The top ten unfounded health scares of 2004: Mercury in seafood causes neurological problems in humans. New York, USA: American Council on Health and Science (ACHS); 2004. http://www.acsh.org/healthissues/newsID.1014/healthissue_detail.asp, accessed 31 Oct. 2005.

Kjellström T. Physical and mental development of children with prenatal exposure to mercury from fish, vol. 3642. Solna, Sweden: National Swedish Environmental Protection Board Report; 1989. 112 pp.

Kudo A, Turner RR. Mercury-contamination of Minamata Bay: historical overview and progress towards recovery. In: Ebinghaus R, Turner RR, Lacerda LDde, Vasiliev O, Salomons W, editors. Mercury contaminated sites. Characterization, risk assessment and remediation. Berlin, Heidelberg, Germany: Springer-Verlag; 1999. p. 143–56.

Lundgren T. Gamla klor-alkalifabriken i Skoghall, Hammarö kommun, Sanering av fabriksområdet, Miljökonsekvensbeskrivning. (Environmental assessment of remediation of the chlor–alkali plant at Skoghall). Linköping, Sweden: Envipro miljöteknik AB; 2001. 36 pp. [in Swedish].

Macdonald RW, Harner T, Fyfe J. Recent climate change in the Arctic and its impact on contaminant pathways and interpretation of temporal trend data. Sci. Total Environ. 2005;342(1–3):5-86.

Max W, Rice DP, Sung H-Y, Michel M. Valuing human life: estimating the present value of lifetime earnings. San Francisco, USA: Institute for Health and Aging; 2000.

Maxson, P. Mercury flows in Europe and the world: The impact of decommissioned chlor–alkali plants. Prepared by Concorde East/West Sprl for the European Commission (Environment Directorate), final report dated February 2004, Brussels, Belgium. http://europa.eu.int/comm/environment/chemicals/mercury/index.htm, accessed 15 March 2004.

Meili M. The coupling of mercury and organic matter in the biogeochemical cycle— towards a mechanistic model for the boreal forest zone. Water Air Soil Pollut 1991;56:333–47.

Meili M. Mercury in lakes and rivers. In: Sigel A, Sigel H, editors. Mercury and its effects on environment and biology. Metal Ions in Biological SystemsNew York: Marcel Dekker Inc.; 1997. p. 21–51.

Meili M. Föroreningshistorik i och utanför Örserumsviken, Västerviks kommun (Contamination history of Örserumsviken). Stockholm University, Sweden: ITM; 2002. http://www.vastervik.com/miljo/orserum/rapport/Meili.pdf, updated April 2002, accessed 15 October 2004 [in Swedish].

Minamata Disease Municipal Museum (Ed.) Minamata Disease—Its History and Lessons—2000. Minamata City, Minamata, Japan, 2000. 64 pp.

Muir D, Braune B, DeMarch B, Norstrom R, Wagemann R, Lockhart L, et al. Spatial and temporal trends and effects of contaminants in the

Canadian Arctic marine ecosystem: a review. Sci Total Environ 1999;230(1–3):83-144.

Murayama, M. Top court finds gov't responsible in Minamata disease suit. Bloomberg. http://quote.bloomberg.com/apps/news?pid=10000101&sid=a8SulqjNMJE4&refer=japan, updated 15 October 2004, accessed 16 Oct. 2004.

National Research Council. Toxicological effects of methylmercury. Washington, DC, USA: National Academy Press; 2000.

Ninomiya T, Imamura K, Kuwahata M, Kindaichi M, Susa M, Ekino S. Reappraisal of somatosensory disorders in methylmercury poisoning. Neurotoxicol Teratol 2005;27(4):643–53.

Norwegian Pollution Control Authority (SFT). 2005. United States the largest mercury source in the North. http://www.sft.no/english/news/utskriftversjon_dbafile13381.html, accessed 15 Oct. 2005.

Oostdam J, Gilman A, Dewailly E, Usher P, Wheatley B, Kuhnlein H, et al. Human health implications of environmental contaminants in Arctic Canada: a review. Sci Total Environ 1999;230(1–3):1-82.

O'Neill, C. A. Mercury, Risk, and Justice. Environmental Law Reporter, News and Analysis, December, 2004. 34 ELR 11070. Environmental Law Institute, Washington, USA. 65 pp.

Pacyna JM, Keeler GJ. Sources of mercury in the Arctic. Water Air Soil Pollut 1995;80:621–32.

Pacyna EG, Pacyna JM. Global emission of mercury from anthropogenic sources in 1995. Water Air Soil Pollut 2002;137:149–65.

Paeth H, Hense A, Glowienka-Hense R, Voss R, Cubasch U. The North Atlantic Oscillation as an indicator for greenhouse-gas induced regional climate change. Clim Dyn 1999;15(12):953–60.

Pirrone N, Keeler GJ, Nriagu JO. Regional differences in worldwide emissions of mercury to the atmosphere. Atmos Environ 1996;30:2981–7.

Pollack, A. 1997. Victims of Mercury-Contaminated Fish Cheer Government Edict, but Scars Remain. http://jcbmac.chem.brown.edu/baird/chem12/readings/nytimes/related/0823japan-mercury.html, updated 1997-08-23, accessed 15 October 2004.

Poulson D. U.S. unloading toxic mercury. Booth/Newhouse Newspapers; 1994. June 23.

Pretty JN, Mason CF, Nedwell DB, Hine RE, Leaf S, Dils R. Environmental costs of freshwater eutrophication in England and Wales. Environ Sci Technol 2003;37(2):201–8.

Prochazkova J, Sterzl I, Kucerova H, Bartova J, Stejskal VDM. The beneficial effect of amalgam replacement on health in patients with autoimmunity. Neuroendocrinol Lett 2004;25(3):211–8.

Projekt Turingen. Projektsammanfattning (Project summary). http://www.turingen.com/ updated 2003-02-10, accessed 10 October 2004 [in Swedish].

Rae D, Graham L. Benefits of reducing mercury in saltwater ecosystems. A case study. Washington, D.C., USA: U.S. Environmental Protection Agency; 2004. 113 pp.

Rein K von, Hylander LD. Experiences from phasing out the use of mercury in Sweden. Regional Environ Change J 2000;1:126–34.

Rice G, Hammitt JK. Economic Valuation of Human Health Benefits of Controlling Mercury Emissions from U.S. Coal-Fired Power Plants. USA: NESCAUM, Northeast States for Coordinated Air Use Management; 2005. 223 pp. www.nescaum.org.

Riget E, Dietz R, Vorkamp K, Johansen P, Muir D. Levels and spatial and temporal trends of contaminants in Greenland biota: an updated review. Sci Total Environ 2004;331(1–3):29–52.

Rothschild RF, Duffy LK. Methylmercury in the hair of subsistence food users in a rural Alaskan village. Alaska Med 2002;44(1):2–7.

Riksbanken, 2004. Currency fixing by commercial banks in Sweden. Monthly average, September 2004. http://www.riksbank.com/upload/Dokument_riksbank/Kat_statistik/mankurs/2004/man0409e.xls, accessed 10 October 2004.

SCB. Cost-of-living Index. Statistics Sweden. http://www.scb.se/snabb/priser/levkostsv.asp, accessed 27 March 2004.

Shimazu-Kohdera T. Mercury and atopic dermatitis: an epidemilogical study of the influence of dental amalgam on the development of atopic dermatitis in children. Skin Res 2000;42(suppl. 22):15–21.

Schroeder WH, Anlauf KG, Barrie LA, Lu JY, Steffen A. Arctic springtime depletion of mercury. Nature 1998;394:331–2.

Schroeder WH, Munthe J. Atmospheric mercury— an overview. Atmos Environ 1998;32:809–22.

Schroeder WH, Steffen A, Scott K, Bender T, Prestbo E, Ebinghaus R, et al. Summary report: first international Arctic atmospheric mercury research workshop. Atmos Environ 2003;37(18):2551–5.

Semkin RG, Mierle G, Neureuther RJ. Hydrochemistry and mercury cycling in a High Arctic watershed. Sci Total Environ 2005;342(1–3):199–221.

Skare I, Engqvist A. Human exposure to mercury and silver released from dental amalgam restorations. Arch Environ Health 1994;49(5):384–94.

Skov H, Christensen JH, Goodsite ME, Heidam NZ, Jensen B, Wåhlin P, et al. Fate of elemental mercury in the Arctic during atmospheric mercury depletion episodes and the load of atmospheric mercury to the Arctic. Environ Sci Technol 2004;38(8):2373–82.

Slemr F, Schuster G, Seiler W. Distribution, speciation and budget of atmospheric mercury. J Atmos Chem 1985;3:407–34.

Slemr F, Brunke E-G, Ebinghaus R, Temme C, Munthe J, Wängberg I, et al. Worldwide trend of atmospheric mercury since 1977. Geophys Res Lett 2003;30(10):23-1–4.

Sørensen N, Murata K, Budtz-Jørgensen E, Weihe P, Grandjean P. Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age. Epidemiology 1999;10:370–5.

SR. Sätt pris på livet — ett program om livets värde (Put a price on the life — a radio programme about the value of life). Vetandets värld, Sveriges Radio, Stockholm, Sweden. 2005. http://www.sr.se/cgi-bin/p1/program/artikel.asp?ProgramID=412&artikel=576967, accessed 5 April 2005 [in Swedish].

Steuerwald U, Weihe P, Jørgensen PJ, Bjerve K, Brock J, Heinzow B, et al. Maternal seafood diet, methylmercury exposure, and neonatal neurologic function. J Pediatr (St. Louis) 2000;136(5):599–605.

St. Louis VL, Sharp MJ, Steffen A, May A, Barker J, Kirk JL, et al. Some sources and sinks of monomethyl and inorganic mercury on Ellesmere Island in the Canadian High Arctic. Environ Sci Technol 2005;39(8):2686–701.

Takeuchi T, Eto K. The pathology of minamata disease. A tragic story of water pollution. Fukuoka, Japan: Kyushu University Press, Inc.; 1999. 292 pp.

Trasande L, Landrigan PJ, Schechter C. Public health and economic consequences of methyl mercury toxicity to the developing brain. Environ Health Perspect 2005;113(5):590–6.

Tsubaki T, Takahashi H, editors. Recent advances in Minamata disease studies. Methylmercury poisoning in Minamata and Niigata, Japan. Tokyo, Japan: Kodansha; 1986. 214 pp.

Tulinius AV. Mercury, dental amalgam fillings and intellectual abilities in Inuit school children in Greenland. Arctic Med Res 1995;54(2):78–81.

UN. Demographic Yearbook 2002. Table 9, Live births and crude birth rates, by urban/rural residence: 1998–2002; Social indicators, Indicators on income and economic activity. United Nations Publications, New York, USA, 2004. http://unstats.un.org/unsd/demographic/products/dyb/dyb2002/Table09.xls, and http://unstats.un.org/unsd/demographic/products/socind/inc-eco.htm, accessed 24 Oct. 2005.

UN-FAO/WHO-JECFA, 2003. Joint FAO/WHO Expert Commitee on Food Additives, Sixty-first meeting, Rome, 10–19 June 2003, Summary and Conclusions. — Food and Agriculture Organization and World Health Organization of the United Nations, JECFA/61/SC. (22 pp.) ftp://ftp.fao.org/es/esn/jecfa/jecfa61sc.pdf, accessed 24 November 2003.

UNEP Chemicals. Global Mercury Assessment. Report no. 54790-01. Geneva, Switzerland, 2002. 258 pp. http://www.chem.unep.ch/mercury/Report/Final%20Assessment%20report.htm, accessed 10 January 2003.

USEPA. Mercury Study, Report to Congress. EPA-452/R-97-003. US Environmental Protection Agency, USA; 1997.

Watts, J. Mercury poisoning of thousands confirmed. The Guardian, October 16, 2001. http://www.guardian.co.uk/print/0%2C3858%2C4278027-103681%2C00.html, accessed 10 October 2004.

Weihe P, Hansen JC, Murata K, Debes F, Jørgensen PJ, Steuerwald U, et al. Neurobehavioral performance of inuit children with increased prenatal exposure to methylmercury. Int J Circumpolar Health 2002;61:41–9.

Zauke GP, Savinov VM, Ritterhoff J, Savinova T. Heavy metals in fish from the Barents Sea (summer 1994). Sci Total Environ 1994;227(1–3):161–73.